GASTROENTEROLOGY

ABSTRACTS AND CITATIONS

NATIONAL INSTITUTE OF ARTHRITIS, METABOLISM, AND DIGESTIVE DISEASES

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
Public Health Service
National Institutes of Health

JANUARY 1978
VOLUME 12, NO. 1
0001 — 0777

Volume 12 No. 1 Abstract and Citation Nos. 0001–0777

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A monthly publication of the
National Institute of Arthritis, Metabolism, and Digestive Diseases

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Bruce H. Kleinstein, Ph.D., J.D., Assistant Director
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for the
National Institute of Arthritis, Metabolism, and Digestive Diseases
pursuant to Contract Number NO1-AM-4-2204
with the PHS, USDHEW

DHEW Publication No. (NIH) 79-66

ISSN 0016-5093

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis, Metabolism and Digestive Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology have accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis, Metabolism, and Digestive Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

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The Secretary of the Department of Health, Education, and Welfare has determined that the publication of this periodical is necessary in the transaction of the public business required by law of this Department. Use of funds for printing this periodical has been approved by the Director of the Office of Management and Budget through April 30, 1981.

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NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *International Standard ISO833, 1974*, are used. The two-letter state abbreviations are based on the United States Postal Service Code.

LANGUAGE ABBREVIATIONS

Afrikaans	Afr.	Georgian	Geo.	Persian	Per.
Albanian	Alb.	German	Ger.	Polish	Pol.
Arabic	Ara.	Greek	Gre.	Portuguese	Por.
Armenian	Arm.	Hausa	Hau.	Pushto	Pus.
Azerbaijani	Aze.	Hebrew	Heb.	Rumanian	Rum.
Belorussian	Bel.	Hindi	Hin.	Russian	Rus.
Bengali	Ben.	Hungarian	Hun.	Serbian	Ser.
Bulgarian	Bul.	Icelandic	Ice.	Slovak	Slo.
Burmese	Bur.	Indonesian	Ind.	Slovene	Slv.
Chinese	Chi.	Interlingua	Int.	Spanish	Spa.
Croatian	Cro.	Italian	Ita.	Swahili	Swa.
Czechoslovakian	Cze.	Japanese	Jpn.	Swedish	Swe.
Danish	Dan.	Kirghiz	Kir.	Tagalog	Tag.
Dutch	Dut.	Korean	Kor.	Tamil	Tam.
English	Eng.	Latin	Lat.	Telugu	Tel.
Esperanto	Esp.	Latvian	Lav.	Thai	Tha.
Estonian	Est.	Lithuanian	Lit.	Turkish	Tur.
Finnish	Fin.	Marathi	Mar.	Ukranian	Ukr.
Flemish	Fle.	Masai	Mas.	Urdu	Urd.
French	Fre.	Multilingual	Mlt.	Vietnamese	Vie.
Gaelic	Gae.	Norwegian	Nor.	Welsh	Wel.

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	ml, µl	milli-, microliter(s)
ADP	adenosine diphosphate	mm	millimeter(s)
AMP	adenosine monophosphate	mOsm	milliosmolar; milliosmeter(s)
amp	ampere	msec	millisecond(s)
ATP	adenosine triphosphate	mV	millivolt(s)
A	angstrom	min	minute(s)
BSP	sulfobromophthalein	MTD	maximum tolerated dose
C	degrees centigrade	NAD	nicotinamide adenine dinucleotide
cal	calories	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter		
cm	centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
dl	deciliter		
DNA	deoxyribonucleic acid	NADPH	reduced nicotinamide adenine dinucleotidephosphate
ED_{50}	median effective dose(s)		
EDTA	ethylenediamine tetraacetic acid	ng	nanogram (10^{-9})
e.g.	for example	nm	nanometer(s)
EEG	electroencephalogram	Pa	ambient pressure
ESR	erythrocyte sedimentation rate	PAS	periodic acid - Schiff
g	gram(s)	pg	picogram (10^{-12})
µg	microgram(s)	pgEq	picogram equivalent(s)
Hz	Hertz	p.o.	orally
hr	hour(s)	ppb	parts per billion
Ig	immunoglobulin(s)	ppm	parts per million
i.m.	intramuscular	r	Roentgen
i.p.	intraperitoneal	RBC	red blood cells (erythrocytes), red blood count
IU	international unit(s)		
i.v.	intravenous	resp.	respectively
kcal	kilocalorie(s)	RNA	ribonucleic acid
kg	kilogram(s)	s.c.	subcutaneous
K_m	Michaelis constant	sec	second(s)
LD_{50}	median lethal dose(s)	SGOT	serum glutamic-oxalacetic transaminase
LDH	lactic acid dehydrogenase		
m	meter(s)	SGPT	serum glutamic-pyruvic transaminase
M	molar; mole(s)		
mM	millimolar; millimole(s)	U	unit(s)
µM	micromolar; micromole(s)	UV	ultraviolet
ma	milliampere(s)	V	volt
mCi, µCi	milli-, microcurie(s)	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mg	milligram(s)	yr	year(s)

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

0001 AN ULTRASTRUCTURAL STUDY OF OSMIOPHILIA IN NORMAL HUMAN OESOPHAGEAL EPITHELIUM. (Eng.) Logan, K. R.; Milne, G.; Hopwood, D. (Ninewells Hosp. and Medical Sch., Dundee, DD2, 1UB, Scotland). *Histochemistry* 53(2):183-187; 1977.

The forming face of the Golgi stack was studied in biopsy specimens of normal human esophagus, using the prolonged osmication technique. The forming face of the Golgi apparatus was demonstrated in the basal and lower prickle cells. These cells also showed osmium deposition in their mitochondria, endoplasmic reticulum, perinuclear cisternae, and lysosomes. The capillary endothelial cells also showed osmium deposition in their Golgi apparatus and endoplasmic reticulum. These studies suggest that the Golgi apparatus may be involved in forming membrane coating granules, similar to those previously described in skin, vagina, and buccal mucosa.

0002 DISTRIBUTION, ONTOGENY AND ULTRASTRUCTURE OF THE MAMMALIAN SECRETIN CELL. (Eng.) Larsson, L. I.; Sundler, F.; Alumets, J.; Hakanson, R.; Schaffalitzky de Muckadell, O. B.; Fahrenkrug, J. (Inst. Medical Biochemistry, Univ. Aarhus, DK-8000 Aarhus C, Denmark). *Cell Tissue Res.* 181(3): 361-368; 1977.

Secretin cells were studied immunocytochemically in adult rats, cats, pigs, and man, and in fetal and young rat tissues. Secretin cells were demonstrated in the duodenum and jejunum of all species. Calculations on the relative frequency of such cells indicate that the bulk of secretin occurs in the jejunum, which supports the view that secretin may be released by physiological stimulants other than hydrochloric acid. Ultrastructural identification of cat and pig secretin cells confirmed their identity with the ultrastructurally defined S cells, and staining experiments revealed that secretin cells were argyrophilic. Secretin cells were detected in the 17-day-old fetal rat duodenum and showed a developmental pattern similar to that displayed by gastrin cells. Secretin may play a role in the early regulation of growth of the fetal gastrointestinal tract.

0003 MORPHOLOGICAL CHANGES IN THE SMALL INTESTINE FOLLOWING JEJUNO-ILEAL SHUNT IN PARENTERALLY FED RATS. (Eng.) Fenyo, G.; Hallberg, D.; Soda, M.; Roos, K. A. (Karolinska sjukhuset, Stockholm, Sweden). *Scand. J. Gastroenterol.* 11(6):635-640; 1976.

The importance of the presence of food in the intestinal lumen during adaptation to diminished intestinal absorptive surface was studied in rats undergoing a jejuno-ileal bypass (85% of the small intestine excluded) and in sham-operated rats. Body weights of sham-operated and shunt-operated rats fed p.o. with pellets were 151 ± 2.8 and 151 ± 22 g, resp., at the time of surgery and 231 ± 4.1 and 156 ± 4.3 g, resp., 2 weeks after operation. Sham- and shunt-operated rats given total parenteral nutrition (TPN) had body weights of 157 ± 1.6 and 161 ± 8.2 g, resp., at the time of operation and 206 ± 7.5 and 192 ± 8.3 g, resp., 2 weeks after operation. The weight differences were significant at 2 weeks after surgery between pellet-fed, sham-, and shunt-operated rats ($p<0.001$) and between pellet fed and TPN rats in both surgical groups. The functioning part of the small intestine of shunt-operated rats was hypertrophic in both p.o. and TPN fed rats in comparison with sham-operated rats with the corresponding nutrition. When intestinal wet and dry weight per 3-cm intestine were determined from five small intestinal segments 2 weeks after operation, both p.o. and TPN nourished shunt-operated rats showed significantly ($p<0.05$ or less in all cases) increased wet and dry weights in all segments except the top of the blind loop where there was a significant decrease ($p<0.001$) for p.o. fed rats and no significant change for TPN nourished rats. Villus height in both p.o. and TPN nourished shunt-operated rats was significantly ($p<0.01$ or less in all cases) increased in all segments but the top of the blind loop (no significant change) compared with the villus height of sham-operated controls. The absolute villus height was significantly ($p<0.05$ or less) greater in p.o. fed rats than in TPN nourished rats in each surgical group. The percentage differences in villus height between shunt- and sham-operated rats were higher in the TPN group than in p.o. fed rats. The results demonstrate that rats with adequate parenteral nutrition 2 weeks after 85% small intestinal bypass develop hypertrophic changes of the intestinal mucosa, indicating the existence of a nonfood-dependent mechanism in the development of adaptive mucosa changes. Nevertheless, the presence of food seems to stimulate quantitatively greater hypertrophic changes.

0004 FUNCTIONAL ALTERATIONS OF LIVER MITOCHONDRIA IN CHRONIC EXPERIMENTAL ALCOHOLISM. (Eng.) Koch, O. R.; Bedetti, C. D.; Gamboni, M. (Facultad de Medicina, Universidad de Buenos Aires, J. E. Uriburu 950, Buenos Aires, Argentina). *Exp. Mol. Pathol.* 25(3):253-262; 1976.

Morphologic and functional alterations of mitochondria obtained from nonfatty livers of male rats fed a super diet (vitamin and lipotrope-enriched) for 4 months which included a 25% sucrose-32% ethanol mixture (group A), were compared with mitochondrial alterations observed in the livers of three control groups of rats. In the control groups, alcohol was isocalorically replaced by sucrose (group B), by fat (group C), or by a basal diet (group D). Livers from group A rats showed fatty changes similar to those seen in livers of the control groups of rats. However, numerous megamitochondria were observed in group A livers; their pre-

ferential hepatocytic lobular location was in midzonal areas, although they were also found in centrilobular hepatocytes. In control livers, the hepatocytes predominantly affected were in periportal areas, and no megamitochondria were observed. Ultrastructurally, group A enlarged mitochondria displayed bizarre shapes. Their cristae were diminished in number, and helical filaments were occasionally observed in dilated cristae and/or in the outer mitochondrial space. The ultrastructural configuration of hepatocytes from control groups B, C, and D was normal. Maximum mitochondrial respiration rate (ng-atoms of oxygen/min/mg protein) in group A rats was significantly lower ($p < 0.001$, 83 ± 27) than that for group B (176 ± 16), group C (163 ± 28), and group D (159 ± 31) rats when succinate (10 mM) was used as substrate. A similar pattern was observed when L-malate (5 mM)-L-glutamate (5 mM) was used as substrate ($p < 0.001$). When β-hydroxybutyrate (5 mM) was used as substrate, the maximal mitochondrial respiration rate of group A rats (45 ± 9) was significantly ($p < 0.05$) lower than that of group B (61 ± 9) and group C (59 ± 12) rats but not significantly different from that of group D rats (57 ± 15). When succinate or malate-glutamate was used as substrate, the coupling of oxidative phosphorylation in group A rats was similar to that of controls, but with β-hydroxybutyrate a small reduction in coupling was noted in group A rats compared with group B and C rats ($p < 0.02$). These results suggest that hepatic mitochondrial respiration is significantly altered when ethanol is supplied to rats at high levels over long periods and when a nutritional basal diet that does not induce fatty liver is used.

0005 ULTRASTRUCTURAL CYTOCHEMISTRY OF THE SECRETORY GRANULES OF THE HAMSTER SUBMANDIBULAR GLAND. (Eng.) Bloom, G. D.; Carlsoo, B.; Danielsson, A.; Wahlin, T. (Dept. Histology, Univ. Umea, S-90187, Umea, Sweden). *Histochemistry* 51(4):261-268; 1977.

The ultrastructure and cytochemistry of the secretory granules of the male hamster submandibular salivary gland were studied. After fixation in glutaraldehyde followed by osmium tetroxide, the granules were seen to exhibit a characteristic bipartite substructure, with an electron lucid crescenteric rim and a more dense central core. Two regions of the granules were visualized in specimens primarily fixed in Millonig's osmium tetroxide or in potassium permanganate. The electron lucid peripheral portion of the membrane bounded secretory granules further displayed a strong positive reaction after staining of ultrathin sections with the periodic acid-chromic acid-(PA-CrA)-silver technique. The strong periodate reactivity of the rim relative to the core suggested a difference in mucin composition of the two granule regions. With the PA-CrA-silver staining technique, a positive reaction was also observed within the Golgi apparatus of the acinar cells.

0006 QUANTITATIVE AGE CHANGES IN THE HISTOLOGICAL STRUCTURE OF HUMAN SUBMANDIBULAR SALIVARY GLANDS. (Eng.) Scott, J. (Univ. Liverpool, Pembroke Place, P.O. Box 147, Liverpool, L69 3BX, England). *Arch. Oral Biol.* 22(3):221-227; 1977.

The nature, extent, and rate of age-related volumetric tissue changes were examined in human submandibular salivary glands. The glands were obtained from a series of 96 necropsies evenly representative of sex and all decades between 16 and 95 yr. With increasing age, the volume of parenchyma steadily lessened ($p < 0.001$) while the proportions of other tissues rose; this effect was due mainly to loss of acinar tissue, as the volume of ducts underwent a gradual net increase with increasing age. There was an acceleration in the rates of volumetric tissue changes in the two final decades of life. A progressive increase in vascular volume was related to increasing levels of degenerative vascular disease that may, through ischemia, bear some causal relation to the ageing atrophy of parenchyma. The rates of ageing changes were similar in each sex.

0007 AFFERENT ACTIVITY OF THE STOMACH VAGAL AND SYMPATHETIC PATHWAYS IN RELATION TO THERMAL EFFECTS ON ITS WALLS. (Rus.) Nozdratchev, A. D.; Pulatova, M. D.; Tersunov, Z. T.; Chubarova, N. I. (Pavlov's Inst. Physiology, Acad. Science USSR, Leningrad, USSR). *Fiziol. Zh. SSSR* 63(6): 896-903; 1977.

0008 ENDOCRINE CELLS OF THE GUT. II. DISTRIBUTION AND THE BASIS FOR ULTRASTRUCTURAL IDENTIFICATION. (Pol.) Mirecka, J. (Z Zakladu Histologii IBM AM, Cracow, Poland). *Przegl. Lek.* 34(4):419-422; 1977.

0009 COMPARATIVE RADIOLOGICAL AND MORPHOLOGICAL STUDY OF THE HUMAN PANCREAS. 2. MORPHOMETRIC INVESTIGATION OF THE HUMAN PANCREATIC DUCT SYSTEM. (Ger.) Schmitz-Moormann, P.; Hein, J.; Thoma, W.; Ihm, P. (Pathologisches Institut der Universitat Marburg, D-3550 Marburg/Lahn, W. Germany). *Anat. Anz.* 141(5):507-511; 1977.

0010 PECULIARITIES OF THE VENOUS BED HISTOLOGY IN THE HEMORRHOIDAL ZONE OF HUMAN RECTUM. (Rus.) Melman, E. P.; Datsun, I. G. (Ivano-Frankovsk Medical Inst., Ivan-Frankovsk, USSR). *Arkh. Anat. Gistol. Embriol.* 73(8):59-65; 1977.

0011 OCCURRENCE OF PSEUDOISOCYANINE-POSITIVE ENDOCRINE CELLS IN THE MIDGUT OF THE HAMSTER (Cricetulus griseus). (Ger.) Hahn von Dorsche, H.; Hartelt, E.; Fehrmann, P.; Krause, R. (Anatomisches Institut der Ernst-Moritz-Arndt-Universitat Greifswald, Friedrich-Loeffler Strasse 23c, DDR-22 Greifswald, E. Germany). *Acta Histochem.* 59(1):168-171; 1977.

0012 CONSTRUCTION OF MILLIGAN AND MORGAN'S ANORECTAL RING. (Spa.) Gomez Oliveros, L.; Forcada Jimenez, M. (Facultad de Medicina, Universidad Complutense de Madrid, Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 50(4):377-390; 1977.

0013 EVOLUTIONARY CHANGES IN THE ESOPHAGEAL LINING OF VERTEBRATES AND MAN IN THE LIGHT OF THE PHYLEMBRYOGENESIS THEORY. (Rus.) Bazhanov, A. N. (Tselinograd Medical Inst., Tselinograd, USSR). *Arkh. Anat. Gistol. Embriol.* 71(10):61-67; 1976.

0014 HISTOLOGICAL AND ULTRASTRUCTURAL STUDIES OF THE ISOLATION OF RAT SMALL INTESTINE EPITHELIAL CELLS. (Ita.) Cusaro, G.; Marchetti, C.; Poggi, P. (Istituto di Fisiologia umana, Universita di Pavia, Pavia, Italy). *Boll. Soc. Ital. Biol. Sper.* 52(5):371-377; 1976.

0015 THE ULTRASTRUCTURE OF ENTEROCYTES OF THE UPPER PART OF THE MOUSE SMALL INTESTINAL VILLI. (Rus.) Parshkov, E. M.; Brodsky, R. A.; Pogudina, N. A. (Inst. Medical Radiology, Acad. Medical Sciences USSR, Obninsk, USSR). *Tsitologiia* 18(11):1393-1394; 1976.

0016 MORPHO-FUNCTIONAL PECULIARITIES OF THE ENDOCRINE CELLS OF THE SMALL INTESTINE IN CHILDREN. (Rus.) Gavalov, S. M.; Korostyshevskaya, I. M.; Vinogradova, M. S.; Yakovleva, V. I. (Inst. Cytology and Genetics, Siberian Branch of USSR Acad. Sciences, Novosibirsk, USSR). *Vopr. Okhr. Materin. Det.* 21(9):23-28; 1976.

See also, 0021, 0074, 0081, 0088, 0118, 0148, 0218, 0223, 0228, 0229, 0232, 0235, 0236, 0242, 0244, 0246, 0248, 0249, 0282, 0681.

ABSORPTION - EXCRETION - TRANSPORT

0017 INCREASE IN HUMAN INTESTINAL PERMEABILITY FOLLOWING INGESTION OF HYPERTONIC SOLUTIONS. (Eng.) Laker, M. F.; Menziew, I. S. (St. Thomas' Hosp. Medical Sch., Lambeth Palace Rd., London SE1 7EH, England). *J. Physiol. (Lond.)* 265(3):881-894; 1977.

A simple oral loading technique was used to investigate the intestinal permeability of 21 healthy adults following the ingestion of lactulose (L) solutions made hypertonic by the addition of several solutes. The subjects were given 100 ml of a solution containing 5 g L and 20, 40, or 60 g sucrose. Some of these subjects were also tested with L solutions containing 30 g glucose, 12 g glycerol, or 22 g mannitol. The different solutions were given to the subjects in varied order, and there was a 3-day interval between tests. After each test, the urine was collected for 5 hr, and the amount of L excreted was quantitated by paper chromatography. The 5-hr L excretions after ingestion of an L solution without sucrose (isotonic control) were 10-30 mg (median, 15). They rose to 11-50 mg (median, 17) after 20 g sucrose, 8-127 mg (19) after 40 g sucrose, and 12-134 mg (33) after 60 g sucrose. Analysis of variance calculations showed a significant regression ($p<0.01$). Eighteen of the subjects received the glucose-containing loads, and they excreted 13-172 mg (median, 41) L. The amount of L excreted over 5 hr from four subjects given the mannitol loads was 8.9, 9.5, 8.9, and 62.5 mg; these same subjects excreted 31.7, 18.8, 17.0, and 78.5 mg L, resp., after the glycerol loads. The responses of individual subjects were reproducible and consistent. Those with high L excretion following hypertonic sucrose loads responded similarly following glucose, mannitol, and glycerol loads; those with low L transfer also behaved consistently. Intestinal permeability to L is enhanced as the solute concentration is increased, probably as a result of the increased L absorption from the intestinal lumen induced by the hypertonic solutions.

0018 EFFECT OF SEROTONIN TREATMENT ON INTESTINAL TRANSPORT IN THE RABBIT. (Eng.) Donowitz, M.; Charney, A. N.; Heffernan, J. M. (Walter Reed Army Inst. Res., Washington, DC 20012). *Am. J. Physiol.* 232(1):E85-E94; 1977.

To determine whether serotonin is an intestinal secretagogue and thus the cause of diarrhea in the carcinoid syndrome, the effect of serotonin on intestinal water and electrolyte transport was evaluated in rabbits. Two weeks of daily s.c. injection of serotonin (52 μmol/kg) suspended in oil resulted in a blood serotonin level elevated to twice that of controls. Intestinal transport was studied *in vivo* by a perfusion technique. Serotonin treatment resulted in ileal secretion and decreased mid-jejunal absorption of water and electrolytes, but did not affect water absorption in the proximal jejunum or colon. Intestinal absorption of D-glucose and the amino acid L-tryptophan and glucose-dependent water and electrolyte absorption were normal in serotonin-treated

animals. Serotonin-induced ileal secretion was reversed by methysergide, a peripheral antagonist of serotonin action. No alterations in intestinal histology or permeability occurred in serotonin-treated animals. Serotonin-induced intestinal secretion was not associated with alterations in the activities of intestinal mucosal adenylate cyclase, cyclic nucleotide phosphodiesterase, or Na-K-ATPase. This rabbit model for serotonin-induced intestinal water and electrolyte secretion has many characteristics similar to those in patients with the carcinoid syndrome and diarrhea. Both exhibit blood serotonin levels elevated to at least twice normal levels, net intestinal secretion or decreased net absorption of water and electrolytes, reversal of net intestinal secretion to net absorption by methysergide treatment, normal intestinal glucose absorption, and normal glucose-dependent intestinal water and electrolyte absorption with conversion by luminal glucose of net intestinal secretion of water into net absorption.

0019 EFFECTS OF VASOACTIVE INTESTINAL PEPTIDE, SECRETIN, AND RELATED PEPTIDES ON RAT COLONIC TRANSPORT AND ADENYLATE CYCLASE ACTIVITY. (Eng.) Waldman, D. B.; Gardner, J. D.; Zfass, A. M.; Makhlouf, G. M. (Medical Coll. Virginia, Richmond, VA 23298). *Gastroenterology* 73(3):518-523; 1977.

Vasoactive intestinal peptide (VIP), secretin, glucagon, gastric inhibitory peptide (GIP), and partial sequences of VIP and secretin were examined for their effect on colonic transport and adenylate cyclase activity in male Sprague-Dawley rats. Net water flow was measured gravimetrically in muscle-stripped, everted sacs of ascending and descending colon. VIP and secretin, but not glucagon or GIP, stimulated colonic secretion. The sensitivity of the colonic mucosa to VIP was high; the threshold (10^{-11} M) and half-maximal dose (10^{-10} M) for the effect of VIP were 1,000 times less than the threshold (10^{-8} M) and half-maximal dose (10^{-7} M) for the effect of secretin. A subthreshold concentration of theophylline (2 mM) increased the submaximal responses to VIP or secretin up to but not beyond the maximal response to VIP alone (4.1 ± 0.2 µl/mg/30 min). The combined effect of secretin and a maximal concentration of VIP was not additive. These results indicate that VIP and secretin share the same receptor sites and/or intermediary mechanisms. VIP_{14-28} acted as a partial agonist with an efficacy of 0.2 and significantly inhibited the response to VIP. $Secretin_{5-27}$ showed no secretory activity and did not inhibit the response to secretin. The lactamimide RMI 12330A (10^{-3} M) abolished the secretory response to VIP and secretin but had no effect on the secretory response to 10 mM theophylline or on control absorptive flow rate. VIP and secretin, but not glucagon or GIP, stimulated adenylate cyclase activity in homogenates of colon mucosa. The lactamimide RMI 12330A (10^{-3} M) inhibited basal adenylate cyclase activity as well as activity stimulated by VIP, secretin, prostaglandin E_1 and 5'-guanylyl imidodiphosphate. The results obtained in colon mucosa conform to a pattern of structural and functional homology exhibited by VIP and secretin in several tissues (fat, liver, and pancreatic acinar cells), which sets them apart from glucagon and GIP. The mode of action of VIP and secretin on colonic secretion appears to involve the adenylate cyclase-cylic AMP system. The abundance of VIP in the colon of many species and the high sensitivity of transport mechanisms to VIP raise the possibility that VIP may play a local (i.e., paracrine and/or neurocrine) role in the regulation of colonic transport.

0020 THE KINETICS OF ETHANOL ABSORPTION AND ELIMINATION IN TWINS AND SUPPLEMENTARY REPETITIVE EXPERIMENTS IN SINGLETON SUBJECTS. (Eng.) Kopun, M.; Propping, P. (Institut fur Zellforschung am Deutschen Krebsforschungszentrum, P.O. Box 10149, D-6900 Heidelberg, W. Germany). *Eur. J. Clin. Pharmacol.* 11(5):337-344; 1977.

The influence of genetic and environmental factors on the metabolism of a single p.o. dose of ethanol (1.2 ml/kg) was analyzed in 19 identical and 21 fraternal healthy, adult, un-selected male twins. The heritability values of the rates of absorption, degradation, and elimination of ethanol were 0.57, 0.41, and 0.46, resp. Environmental factors, such as daily alcohol intake and smoking, increased the rate of elimination of blood alcohol. Intrasubject variation in ethanol metabolism was studied by repeated tests in 11 male volunteers at intervals of at least 2 months, under the same conditions as in the twins; the coefficient of variation for parameters of metabolism was about 8%. The results demonstrate both genetic control of ethanol absorption, degradation, and elimination and the appreciable influence of environmental factors. The almost total genetic control of ethanol metabolism, which was postulated previously, could not be confirmed.

0021 ALTERATIONS IN THE ENDOPLASMIC RETICULUM AND GOLGI COMPLEX OF INTESTINAL EPITHELIAL CELLS DURING FAT ABSORPTION AND AFTER TERMINATION OF THIS PROCESS: A MORPHOLOGICAL AND MORPHOMETRIC STUDY. (Eng.) Friedman, H. I.; Cardell, R. R. (Dept. Surgery, Letterman Army Inst. Res., Presidio of San Francisco, CA 94129). *Anat. Rec.* 188(1):77-102; 1977.

The morphological changes in the endoplasmic reticulum (ER) and Golgi complex during fat absorption and at successive time intervals after the termination of fat absorption were studied in male rats. Following the administration of corn oil by intubation, the smooth endoplasmic reticulum (SER) was seen to proliferate, and numerous vesicles contained reesterified lipid. The rough endoplasmic reticulum (RER) decreased, and 1 hr after fat administration, the RER only occupied a narrow zone interposed between the Golgi complex and the SER region. These findings were confirmed by morphometric analysis; the RER decreased from approximately 2.4 to 1.2 μ/μ^2 cytoplasm, while the SER increased from approximately 3.7 to 4.8 μ/μ^2 cytoplasm, from the fasted to the

fat-fed state ($p<0.01$). The Golgi complex was seen to accumulate lipid in the Golgi vacuoles after fat administration. Lipid droplets did not occur in saccules near the forming face of the Golgi complex, and as the absorptive process continued, the length and number of Golgi saccules decreased significantly (by 0.74 μ of membrane/μ^2 cytoplasm) from the fasted state to 60 min after fat administration ($p<0.01$). The Golgi vacuoles increased by 0.32 μ of membrane/μ^2 cytoplasm ($p<0.01$). After the termination of fat absorption, using a saline flush, the RER progressively increased, and the quantity of SER lipid-containing vesicles decreased. The Golgi vacuoles decreased in number and size, while the saccules increased in length and number after the termination of fat absorption. The results support the suggestion that during fat absorption, membrane synthesis by the RER is insufficient to accomodate membrane utilization and intraconversion, resulting in a decrease of both ER and Golgi complex components. Following the termination of fat absorption, the cell is able to replace previously depleted components of the ER and Golgi complex and regain the full membrane complement of the fasted state. Replenishment of cellular membranes may result from a continued synthesis of new membranes by the RER, which eventually exceeds the amount membrane utilized during lipid transport.

0022 IRON ABSORPTION IN THE RAT: THE SEARCH FOR POSSIBLE INTESTINAL MUCOSAL CARRIERS. (Eng.) Halliday, J. W.; Powell, L. W.; Mack, U. (Royal Brisbane Hosp., Brisbane 4029, Australia). *Br. J. Haematol.* 34(2):237-250; 1976.

The biological relevance of four iron-containing fractions previously detected in rat intestinal mucosal cells was studied. The distribution of iron in these fractions, obtained by chromatography on Sepharose 6B, was examined after *in vivo* and *in vitro* incubation of mucosal cells with ^{59}Fe. In addition, the effects of phenobarbitone, cycloheximide, iron-deficiency, and iron-loading on the uptake and distribution of iron within the four mucosal cell fractions was studied. The iron in fraction I was mostly bound to intracellular membrane particles. Fraction II was shown to be ferritin. Fraction III contained some transferrin and also a protein of molecular weight similar to transferrin, but which was not precipitable by antitransferrin antiserum. Under all experimental conditions (normal, iron-loaded, iron-deficient, phenobarbitone- and cycloheximide-treated animals) fraction I contained less ^{59}Fe in the *in vivo* than in the *in vitro* studies. In iron deficiency, the ^{59}Fe/protein ratio in fractions I and III was markedly increased, especially *in vivo*, and fraction II (ferritin) was not detectable either by agar gel diffusion or polyacrylamide gel electrophoresis. In iron-loaded animals, the ^{59}Fe/protein ratio was increased in fraction II. Cycloheximide treatment resulted in a decrease in the ^{59}Fe content of fraction I, but not in the other fractions. Phenobarbitone treatment produced no detectable change in any fraction. Quantitative differences between the *in vivo* and *in vitro* studies combined with the results of "chaser" experiments suggested that, in addition to ferritin, at least two fractions (I and III) were involved in the process of iron absorption by the mucosal cells.

0023 Na AND Cl TRANSPORT AND SHORT-CIRCUIT CURRENT IN RABBIT ILEUM. (Eng.) Tai, Y. H.; Desjeux, J. F.; Danisi, G.; Curran, P. F. (Walter Reed Army Medical Center, Washington, DC 20012). *J. Membr. Biol.* 31(1/2):189-208; 1977.

Na and Cl fluxes and short-circuit current (I_{sc}) in rabbit ileum were studied as a function of ionic concentrations in HCO_3-free solutions. Both net Na flux (J^{Na}_{net}) and I_{sc} showed similar saturation functions of [Na] at fixed [Cl]. They showed no significant difference between 0 and 112 mM Na, but at 140 mM Na I_{sc} was significantly greater than the J^{Na}_{net}. Net Cl transport (secretion) was observed only at 140 mM Na and was approximately equivalent to the difference between the I_{sc} and J^{Na}_{net}. The transcellular mucosa-to-serosa Na fluxes measured at 140 and 70 mM Na did not differ significantly from the corresponding I_{sc}. The net Cl flux varied with [Cl] at fixed [Na], but I_{sc} was virtually not affected by [Cl]. These results suggest that the absorptive Na transport process is electrogenic and responsible for the I_{sc} and that the secretory fluxes of Na and Cl are coupled, require high [Na], vary with [Cl], and do not contribute to I_{sc}. A K-free solution abolished the I_{sc} after a prolonged lag. The intracellular Na and K concentrations did not vary significantly with anion concentration. All Cl-containing solutions gave approximately the same intracellular Cl concentration, but the Cl-free solutions gave about 10 mM inexchangeable or slowly exchangeable Cl.

0024 INHIBITION OF ION TRANSPORT BY BILE SALTS IN CANINE GASTRIC MUCOSA. (Eng.) Kuo, Y. J.; Shanbour, L. L. (Univ. Texas Medical Sch., Houston, TX 77025). *Am. J. Physiol.* 231(5):1433-1437; 1976.

The effect of bile salts on ion transport was studied in *in vivo* and *in vitro* canine stomach preparations. The instillation of 5, 10, and 20 mM of bile salts in *N*-tris(hydroxymethyl)methyl-2-aminoethane sulfonic acid buffered to pH 7.4 by NaOH into the nonsecreting stomach *in vivo* caused a progressive decrease in electrical potential difference (PD) and an increase in electrical resistance (R). The rate of acid secretion, determined by the pH-stat method in the histamine-stimulated stomach, decreased with 5 and 20 mM of bile salts. Mucosal ATP content of the nonsecreting or secreting stomach was reduced by bile salts. *In vitro* flux studies demonstrated that within the first hour after 1 mM of bile salts were added to the mucosal side of the chamber, PD decreased, R increased, and net sodium transport decreased. In the second hour, unidirectional fluxes of sodium increased, indicating an increase in permeability of the gastric mucosa to sodium. These results

demonstrate that the initial action of bile salts is inhibition of ion transport, which is followed by an increase in permeability.

0025 ABSORPTION OF AMINO PENICILLINS FROM EVERTED RAT INTESTINE. (Eng.) Dixon, C.; Mizen, L. W. (Beecham Pharmaceuticals, Brockham Park, Betchworth, Surrey, RH3 7AJ, England). *J. Physiol. (Lond.)* 269(3):549-559; 1977.

The absorption of the amino penicillins amoxycillin, ampicillin, cyclacillin, BRL 11792, BRL 10351, BRL 8033, BRL 8035, and BRL 7116 was studied in rat jejunum, using an *in vitro* everted gut sac method to identify the involvement of any specialized transport mechanisms in their absorption across the intestine. Only cyclacillin (1-amino-cyclohexyl penicillin) was shown to be actively transported. The other penicillins appeared to diffuse passively across the intestine. Cyclacillin was found to concentrate against a gradient at 37 C but not at 19 C. Transport of cyclacillin across the mucosal membrane was saturated at mucosal concentrations greater than 1,000 µg/ml. The rate of the forward flux of cyclacillin was many times that of its back flux, the latter being detectable only after 30 min. No relationship between the active transport of cyclacillin and that of amino acids could be demonstrated.

0026 ENERGY-RICH PHOSPHATES AND TRANSINTESTINAL TRANSPORT IN RAT INTESTINE INCUBATED IN VITRO AT DIFFERENT TEMPERATURES. (Eng.) Faelli, A.; Esposito, G.; Capraro, V. (Istituto di Fisiologia Generale e di Chimica Biologica, State Univ. Milan, Milan, Italy). *Biochim. Biophys. Acta* 455(3):759-766; 1976.

Tissue levels of ATP, ADP, and AMP were measured in everted rat jejunum incubated *in vitro* at 28 and 38 C for 1 hr and were compared with levels observed *in vivo* in unperfused (control) jejunum to investigate factors affecting intestinal fluid transport *in vitro*. Intestinal fluid transport values of 2.20 ± 0.24 and 0.98 ± 0.24 mg/g total dry weight intestine/hr were measured for intestines perfused *in vitro* for 1 hr at 28 and 38 C, resp.; these differences were significant ($p<0.01$). A comparison of wet/dry weight ratios of *in vitro* perfused intestinal mucosa scrapes and the remaining submucosa (at both incubation temperatures) with wet/dry weight ratios of *in vivo* unperfused control mucosa and submucosa revealed mucosal swelling under *in vitro* conditions at both temperatures ($p<0.01$ at 28 C and $p<0.001$ at 38 C); swelling was greater at 38 C. Additional fluid transport measurements made every 20 min during *in vitro* intestinal perfusion for 1 hr at 28 and 38 C revealed that fluid transport was constant throughout the 1-hr perfusion period at 28 C but progressively decreased with time at 38 C. ATP levels in total intestine and mucosal scrapes at the beginning of perfusion at 28 C were reduced from control (*in vivo*) values of 7.6 ± 0.6 and 6.7 ± 0.7 µmol/g, resp., to 3.7 ± 0.4 and 4.4 ± 0.3 µmol/g, resp. After 1 hr of perfusion at 28 C, these levels remained fairly constant, being 3.8 ± 0.3 and 3.8 ± 0.4 µmol/g, resp. However, ATP levels in total intestine and mucosal scrapes perfused at 38 C fell from values of 2.4 ± 0.4 and 1.9 ± 0.5 µmol/g, resp., recorded at the beginning of perfusion, to levels of 0.8 ± 0.1 and 0.4 ± 0.1 µmol/g, resp., after 1 hr of perfusion. A similar trend was seen for ADP. AMP levels under the above conditions did not vary appreciably. These results indicate that fluid transport and ATP content of *in vitro* perfused intestine are directly related. Transport activity, which is lower at 38 C than at 28 C, appears to be due to a low availability of energy-rich phosphates.

0027 ROLE OF 1,25-DIHYDROXYVITAMIN D_3 ON INTESTINAL PHOSPHATE ABSORPTION IN RATS WITH A NORMAL VITAMIN D SUPPLY. (Eng.) Rizzoli, R.; Fleisch, H.; Bonjour, J. P. (Dept. Pathophysiology, Univ. Berne, 3010 Berne, Switzerland). *J. Clin. Invest.* 60(3):639-647; 1977.

The question of whether changes in 1,25-dihydroxyvitamin D_3 [1,25-$(OH)_2D_3$] production can influence intestinal phosphorus transport in animals with a normal supply of vitamin D was investigated. The intestinal P absorption was evaluated in rats using both the *in situ* duodenal loop technique and the determination of the overall gastrointestinal absorption under three conditions known to influence the production of 1,25-$(OH)_2D_3$: (a) variation in dietary P, (b) thyroparathyroidectomy (TPTX) with or without administration of parathyroid hormone (PTH), and (c) treatment with disodium ethane-1-hydroxy-1,1-diphosphonate (EHDP). In all circumstances, changes in duodenal absorption paralleled the changes in the overall fractional absorption. Lowering dietary P stimulated P absorption. TPTX decreased P absorption. This effect was corrected either by the administration of PTH or by the administration of 1,25-$(OH)_2D_3$. EHDP, when given at a dose known to inhibit 1,25-$(OH)_2D_3$ formation, decreased the duodenal P absorption in both intact and TPTX animals. This effect was corrected by 1,25-$(OH)_2D_3$. In the TPTX-EHDP-treated animals, the administration of PTH did not rectify the low duodenal P absorption. These results support the thesis that, in rats with normal vitamin D supply, variations in the endogenous production of 1,25-$(OH)_2D_3$ change the rate of P absorption. However, these changes are in such magnitude that they are of relatively small importance when compared to the effect of variation in the dietary intake of P. These results also strongly suggest that the action of PTH on duodenal P transport is mediated by its effect on 1,25-$(OH)_2D_3$ production, inasmuch as the effect of the hormone is abolished after blocking the renal 1-hydroxylation with EHDP.

0028 CYCLIC AMP-DEPENDENT AMINO ACID UPTAKE IN INTESTINE--THE IMPORTANCE OF β-ADRENERGIC AGONISTS. (Eng.) Kinzie, J. L.; Grimme, N. L.; Alpers, D. H. (Washington Univ. Sch. Medicine, St. Louis, MO 63110). *Biochem. Pharmacol.* 25(24):2727-2731; 1976.

Cyclic AMP (cAMP)-dependent stimulation of neutral and dibasic amino acid uptake by intestinal mucosa was investigated, using everted rat intestinal rings. β-Adrenergic receptor sites on the mucosal cell itself were implicated, since epinephrine and isoproterenol stimulated uptake *in vitro*, and this uptake was blocked by propranolol. Glucagon, vasopressin, and norepinephrine had no effect on tissue cAMP levels or on amino acid uptake, but cholera toxin given *in vivo* increased both parameters tested *in vitro*. Theophylline and isoproterenol caused an increase in cAMP levels in the villus tip cells, where these drugs also stimulated amino acid uptake. Neither drug increased cAMP in crypt cells. Thus, the cAMP-stimulated uptake of amino acids *in vitro* is a function of the villus tip cells. Moreover, when metabolic products, such as cAMP, are measured in whole intestinal mucosa, it cannot be assumed that the change is uniform throughout the tissue, but may involve only one type of cell on the intestinal villus.

0029 PROTEIN, DIPEPTIDE, AND AMINO ACID ABSORPTION IN THE YOUNG OF PROTEIN-DEPRIVED RATS. (Eng.) Zeman, F. J.; Fratzke, M. L. (Dept. Nutrition, Univ. California, Davis, CA 95616). *Pediatr. Res.* 11(9):972-977; 1977.

The quantitative effect of prenatal protein deficiency on protein, dipeptide, and amino acid transport was studied in Sprague-Dawley rats, and experiments were carried out to determine whether alterations in the absorption of these substances could be correlated with a decreased population of enterocytes or their reduced functional ability. Absorption was studied *in vivo* by infusing intestines of newborn and postnatal young of female rats (fed diets containing 4% or 24% casein during pregnancy) with 50 mM ^{14}C-labeled 1-aminocyclopentane-1-carboxylic acid (ACC), α-aminoisobutyric acid (AIB), glycyl-1-L-leucine (GL), or 8% ^{3}H-histidine-labeled casein in 5% glucose and physiologic saline. Casein absorption was significantly reduced per absorptive cell in prenatally protein-deprived (PPD) young at birth ($p<0.05$), but there were no differences at 8 days of age. AIB absorption and retention were decreased in PPD newborn young compared to controls, whether expressed as a total, on the basis of body weight, or per enterocyte. At 12 days, total retention and absorption were reduced in PPD young, but all other differences no longer existed. ACC absorption was reduced in PPD young, compared to controls, regardless of the means of expression. Retention of ACC per enterocyte was increased at birth compared to controls. These differences had disappeared by 8 days. GL retention was increased in PPD young compared to controls at birth, but not at 8 days. The data on casein absorption indicate that newborn PPD young are handicapped at some point in the processes of amino acid absorption and digestion and protein absorption. The high retention of dipeptide per enterocyte may be the consequence of a decreased ability of the enterocytes of PPD young to hydrolyze the dipeptide or may be the result of increased incorporation of these materials into mucosal protein. There is apparently a marked difference between the effects of intestinal absorption of prenatal protein deprivation and those of postnatal malnutrition. It is suggested that the competence of the enterocytes is reduced in newborn PPD young, whereas animals whose prenatal nutrition was normal begin life with a larger complement of fully differentiated absorptive cells and might therefore be capable of greater adaptation to the nutritional deficit.

0030 VITAMIN K_2 COLONIC AND ILEAL IN VIVO ABSORPTION: BILE, FATTY ACIDS, AND pH EFFECTS ON TRANSPORT. (Eng.) Hollander, D.; Rim, E.; Ruble, P. E., Jr. (Dept. Medicine, Wayne State Univ., Detroit, MI 48201). *Am. J. Physiol.* 233(2): E124-E129; 1977.

Colonic and ileal absorption of vitamin K_2 ([2-methyl-^{3}H]menaquinone-9) was investigated in the conscious rat, and the effects of the vitamin concentration, the thickness of the unstirred water layer, the concomitant presence of fatty acids, the intraluminal pH, and the intraluminal sodium taurocholate concentration on absorption were studied. When the absorption rate was plotted against the perfusate concentration, a linear relationship was found between these two parameters in the ileum and colon. The absorption rate of menaquinone by the ileum was increased as the bile salt concentration, degree of unsaturation of the added long-chain fatty acids, hydrogen ion concentration, and perfusate flow rates were increased. Colonic menaquinone absorption decreased as the bile salt concentration was increased. Menaquinone colonic absorption increased as the pH decreased, but no change was noted as the perfusion rate was increased. The present observations *in vivo*, coupled with prior observations *in vitro*, indicate that the absorption of menaquinone by the ileum and colon occurs by a passive diffusion process that is modified by variations in the perfusate bile salt concentration, the presence of unsaturated fatty acids, and the perfusate pH. The results indicate that the mammalian colon and terminal ileum can provide a constant source of vitamin K to aid hemostasis despite episodic lack of dietary vitamin K.

0031 SITE OF ACTION OF ACETYLCHOLINE IN REGULATING INTESTINAL EPITHELIAL ION TRANSPORT IN THE RAT [Abstract]. (Eng.) Browning, J. G.; Hardcastle, J.; Hardcastle, P. T.; Redfern, J. S. (Dept. Physiology, Sheffield Univ., Sheffield, England). *J. Physiol. (Lond.)* 270(1):78P-79P; 1977.

0032 COMPARATIVE METABOLISM OF BENORYLATE AND AN EQUIVALENT MIXTURE OF ASPIRIN AND PARACETAMOL IN NEONATE AND ADULT RABBITS. (Eng.) Davison, C.; Dorrbecker, B. R.; Edelson, J. (Sterling-Winthrop Res. Inst., Rensselaer, NY 12144). *Xenobiotica* 7(9):561-571; 1977.

0033 STUDIES ON THE ABSORPTION OF THIAMINE, RIBOFLAVIN AND PYRIDOXINE IN VITRO. (Eng.)

Meinen, M.; Aeppli, R.; Rehner, G. (Institut fur Ernahrungswissenschaft I, Justus Liebig-Universitat, Wilhelmstrasse 20, D-6300 Giessen, W. Germany). *Nutr. Metab.* 21(Suppl. 1):264-266; 1977.

0034 INFLUENCE OF GUMS ON INTESTINAL ABSORPTION. (Eng.) Forster, H.; Hoos, I. (Zentrum der Biologischen Chemie, Universitat Frankfurt, Theodor-Stern-Kai 7, D-6000 Frankfurt/Main, W. Germany). *Nutr. Metab.* 21(Suppl. 1):262-264; 1977.

0035 EFFECT OF SODIUM ON THE ACTIVE TRANSPORT OF SUGARS IN THE RAT. (Eng.) Beyer, R.; Forster, H. (Zentrum der Biologischen Chemie, Universitat Frankfurt, Theodor-Stern-Kai 7, D-6000 Frankfurt/Main, W. Germany). *Nutr. Metab.* 21(Suppl. 1): 259-261; 1977.

0036 INFLUENCE OF THE IMMUNE RESPONSE ON THE ABSORPTION OF PROTEIN FROM THE GUT. (Eng.) Seifert, J.; Ring, J.; Steininger, J.; Brendel, W. (Dept. Surgery, Univ. Munich, Nussbaumstrasse 20, D-8000 Munchen 8, W. Germany). *Nutr. Metab.* 21 (Suppl. 1):256-258; 1977.

0037 INFLUENCE OF FOOD AND DIET ON GASTRO-INTESTINAL DRUG ABSORPTION: A REVIEW. (Eng.) Welling, P. G. (Sch. Pharmacy, Univ. Wisconsin, Madison, WI 53706). *J. Pharmacokinet. Biopharm.* 5(4):291-334; 1977.

0038 CHANGES IN THE ORAL ABSORPTION CHARACTERISTICS IN MAN OF DIPOTASSIUM CLORAZEPATE AT NORMAL AND ELEVATED GASTRIC pH. (Eng.) Abruzzo, C. W.; Macasieb, T.; Weinfeld, R.; Rider, J. A.; Kaplan, S. A. (Hoffmann-La Roche Inc., Nutley, NJ 07110). *J. Pharmacokinet. Biopharm.* 5(4):377-390; 1977.

0039 INFLUENCE OF IONIC ENVIRONMENT ON INTESTINAL OXYGEN CONSUMPTION. (Eng.) Jackson, M. J.; Kutcher, L. M. (George Washington Univ. Medical Center, Washington, DC 20037). *Experientia* 33(8):1061; 1977.

0040 DIFFERENT RESPONSE OF THE BIRD AND MAMMAL INTESTINAL ACTIVE TRANSPORT TO CALCIUM. (Eng.) Bolufer, J.; Larralde, J. (Departamento de Fisiologia Animal, Universidad de Navarra, Pamplona, Spain). *Comp. Biochem. Physiol.* 58(1A):75-79; 1977.

0041 EVERTED RAT INTESTINAL SACS AS AN IN VITRO MODEL FOR ASSESSING ABSORPTIVITY OF NEW DRUGS. (Eng.) Chowhan, Z. T.; Amaro, A. A. (Syntex Res., Palo Alto, CA 94304). *J. Pharm. Sci.* 66(9):1249-1253; 1977.

0042 ABSORPTION AND METABOLISM OF CHLORINATED FATTY ACIDS AND TRIGLYCERIDES IN RATS. (Eng.) Cunningham, H. M.; Lawrence, G. A. (Health and Welfare Canada, Tunney's Pasture, Ottawa, Canada). *Food Cosmet. Toxicol.* 15(2):101-103; 1977.

0043 ABSORPTION STUDIES WITH THE ANTI-DIARRHOEAL AGENT ETHACRIDINE LACTATE IN LABORATORY ANIMALS AND MAN. (Eng.) Rising, T. J.; Fromson, J. M.; McEwen, J.; Johnson, P. (Hoechst Pharmaceutical Res. Lab., Walton Manor, Milton Keynes, Buckinghamshire, England). *Arzneim. Forsch.* 27(I, 4):872-878; 1977.

0044 SEGMENTAL DIFFERENCE IN TRANSMUCOSAL FLUID MOVEMENT AND GLUCOSE EFFECT ON DRUG ABSORPTION FROM RAT GASTROINTESTINAL TRACT. (Eng.) Kitazawa, S.; Ito, H.; Sezaki, H. (Kyoto Univ. Hosp., Kawara-cho Shogoin, Sakyo-ku, Kyoto, Japan). *Chem. Pharm. Bull.* 25(1):19-28; 1977.

0045 GI DRUG ABSORPTION IN RATS EXPOSED TO COBALT-60 γ-RADIATION III: IN SITU INTESTINAL ABSORPTION. (Eng.) Brady, M. E.; Hayton, W. L. (Coll. Pharmacy, Univ. Cincinnati, Cincinnati, OH 45221). *J. Pharm. Sci.* 66(5):680-683; 1977.

0046 RESORPTION OF VITAMIN B_1--XII. CHANGES IN THE RESORPTION AND THE PHOSPHORYLATION OF THIAMINE IN RATS IN RELATION TO AGE. (Eng.) Lazarov, J. (Inst. Animal Breeding, Kostinbrod, Bulgaria). *Exp. Gerontol.* 12(1/2):75-79; 1977.

0047 FACTORS INFLUENCING ABSORPTION AND EXCRETION OF DRUGS. VI. EFFECT OF SODIUM CHOLATE ON IN SITU RAT INTESTINAL ABSORPTION OF BUFORMIN. (Eng.) Kojima, S.; Hamada, C.; Tanaka, R. (Faculty Pharmaceutical Sciences, Kumamoto Univ., 5-1, Oehon-machi, Kumamoto, 862, Japan). *Chem. Pharm. Bull.* 25(6):1243-1248; 1977.

0048 SMALL INTESTINAL ABSORPTION OF METHYLENE BLUE IN RATS, GUINEA PIGS AND RABBITS. (Eng.) Watanabe, J.; Mori, K. (Faculty Pharmaceutical Sciences, Nagoya City Univ., 3-1, Tanabe-dori, Mizuho-ku, Nagoya, 467, Japan). *Chem. Pharm. Bull.* 25(6):1194-1201; 1977.

0049 ENHANCEMENT OF INTESTINAL SUGAR TRANSPORT BY RATS FED SUCROSE AS COMPARED TO STARCH. (Eng.) Reiser, S.; Hallfrisch, J.; Putney, J.; Lev, F. (Nutrition Inst., U.S. Dept. Agriculture, Beltsville, MD 20705). *Nutr. Metab.* 20(6):461-470; 1976.

0050 ENTEROHEPATIC CIRCULATION OF SODIUM SULISATIN AND ITS EFFECTS ON GLUCOSE ABSORPTION IN THE RAT. (Eng.) Moreto, M.; Gonalons, E.; Mylonakis, N.; Giraldez, A.; Torralba, A. (Facultad de Farmacia, Zona Universitaria de Pedralbes, Barcelona [14], Spain). *J. Pharm. Pharmacol.* 29(7):446-448; 1977.

See also, 0090, 0115, 0230, 0244, 0273, 0275, 0289, 0290, 0423.

MOTILITY

0051 LONGITUDINAL FORCE AND STRESS OF RAT'S ESOPHAGUS: AGE-RELATED CHANGES. (Eng.) Zabinski, M. P.; Biancani, P. (Dept. Internal Medicine, Yale Univ., New Haven, CT 06510). *Am. J. Physiol.* 232(6):E580-E583; 1977.

The esophageal length, force, and stress characteristics were studied in rats, and the effect of age on these parameters was investigated. The longitudinal force-length relationship was studied *in vitro* in three age groups: 1, 3, and 12 months. The length of maximum force development (MFD) occurred at 1.4-1.5 times the *in vivo* length for all age groups. The active force developed at MFD increased markedly with age. The difference in the active forces in the 3-month and 12-month age groups is due to differences in cross section, because the active stress of the esophagus in the longitudinal direction was found to be approximately equal for the two age groups. The active stress in the 1-month-old rats was lower than in the 3-month-old rats, suggesting an increased contractility of the esophagus with age in this period of development.

0052 ESOPHAGEAL PERISTALTIC FORCE IN MAN: RESPONSE TO MECHANICAL AND PHARMACOLOGICAL ALTERATIONS. (Eng.) Schoen, H. J.; Morris, D. W.; Cohen, S. (Hosp. Univ. Pennsylvania, Philadelphia, PA 19104). *Am. J. Dig. Dis.* 22(7):589-597; 1977.

Studies were carried out in eight healthy subjects to quantify human esophageal peristaltic force, to determine the factors that alter this force, and to evaluate the relationship between force and intraluminal pressure. Force was measured using a mercury-in-Silastic strain gauge. Simultaneous pressures were obtained through constantly infused open-tipped catheters. On separate days, a sphere of either 6.0-, 9.0-, or 13.0-mm diameter was attached to the strain gauge, and measurements were obtained at three esophageal levels. Studies were done (a) with the subject at rest, (b) while performing a Valsalva maneuver, (c) during the administration of bethanechol (0.1 mg/kg, s.c.), and (d) after atropine (0.01 mg/kg, i.v.). Peristaltic force increased directly with sphere size but inversely with distance from the lower esophageal sphincter. Intraluminal pressure did not change significantly with sphere size or at different levels of the esophagus. A Valsalva maneuver increased force and pressure significantly only in the distal esophagus. Bethanechol had no significant effect on force at any esophageal level, but atropine decreased force at each esophageal level and with each sphere size. The distal esophageal response to a Valsalva maneuver was lost following atropine. Intraluminal pressure was a good index of peristaltic force, providing sphere size and esophageal level remained unaltered. These studies suggest that esophageal peristaltic force may be altered by bolus size, extrinsic maneuvers that alter intraluminal pressure (Valsalva maneuver), and certain pharmacological agents.

0053 CALCIUM DEPENDENCE OF NEUROMUSCULAR EVENTS IN ESOPHAGEAL SMOOTH MUSCLE OF THE OPOSSUM. (Eng.) de Carle, D. J.; Christensen, J.; Szabo, A. C.; Templeman, D. C.; McKinley, D. R. (Univ. Hosp., Iowa City, IA 52242). *Am. J. Physiol.* 232(6):E547-E552; 1977.

The relationship between extracellular calcium concentration and resting active tension and nerve-mediated events in the sphincteric or esophagogastric junctional region was examined. Intramural nerves in smooth muscle strips from opossum esophagus were stimulated electrically to cause contractions of longitudinal body muscle, off responses of transverse body muscle, and relaxations of sphincter muscle. Krebs solution was modified by calcium removal, by strontium substitution for calcium, by adding magnesium (2.4-22.4 mEq/l), and by adding nitroprusside (10^{-7}-10^{-5} M). Longitudinal muscle contractions were abolished by calcium removal and by excess magnesium; they were unaffected by nitroprusside; strontium could not replace calcium. Off responses were abolished by calcium removal and by excess magnesium; they were depressed by nitroprusside; strontium

effectively replaced calcium. Resting active tension in sphincter strips was partly reduced by calcium removal and by excess magnesium; it was abolished by nitroprusside; strontium could not replace calcium. Relaxations in sphincter strips were unaffected by all experimental conditions. Longitudinal contractions, off responses, and resting active tension of the sphincter represent different kinds of calcium activation of muscle. The excitatory nerves (which are cholinergic) in longitudinal muscle are calcium dependent, whereas the nerves that produce off responses may not be. The nerves that relax the sphincter are not calcium dependent.

0054 THE INFLUENCE OF THE CALCIUM ANTAGONIST FENDILINE ON TONE AND MOTILITY OF THE GUINEA PIG GUT SMOOTH MUSCLE AND THE cAMP AND cGMP CONCENTRATIONS OF THE ISOLATED TERMINAL ILEUM. (Eng.) Sprugel, W.; Mitznegg, P.; Heim, F. (Pharmakologischen Instituts der Universitat Erlangen-Nurnberg, 8520 Erlangen, W. Germany). *Arzneim. Forsch.* 27(3):571-574; 1977.

The effect of *N*-(2-benzhydryl-ethyl)-*N*-(1-phenyl-ethyl)-amine hydrochloride (fendiline) on smooth muscle tone was studied in the guinea pig gastrointestinal tract, and the interactions between this drug and calcium, cyclic AMP, cyclic GMP, and theophylline in the terminal ileum were examined. Fendiline decreased the smooth muscle tone elevated by K^+-induced depolarization. This effect was antagonized by the addition of extra-Ca^{++}. The muscle relaxation was dose-dependent and amounted to 45-90% after 1-5 µg/ml fendiline. Proportionally to this effect, the tissue concentration in cyclic GMP was decreased whereas cyclic AMP remained unchanged. After 54 µg/ml theophylline, the cyclic AMP level in the terminal ileum was increased significantly, whereas cyclic GMP did not change. Theophylline had no influence on the relaxing effect of 1 µg/ml fendiline. By contrast, the increase in cyclic AMP after theophylline was prevented by fendiline. These findings are explained by the antagonistic effect of fendiline to Ca^{++}, which activates the guanylate cyclase and inhibits the adenylate cyclase. Fendiline seems to prevent the binding of theophylline to guinea pig ileal phosphodiesterase.

0055 SHORT CIRCUIT CURRENT AND TOTAL CONDUCTANCE MEASUREMENTS ON RAT ILEUM. (Eng.) Emilio, M. G.; De Jesus, C. H. (Centro de Biologia, Instituto Gulbenkian de Ciencia, Oeiras, Portugal). *J. Physiol. (Lond.)* 270(1):209-222; 1977.

To determine the effects of different sugar molecules (D-glucose, 3-*O*-methyl-D-glucose, and 3-deoxy-3-fluor-D-glucose) on short circuit current and total conductance of the intestine, these measurements were made in preparations of rat terminal ileum, using either the intact tissue or the isolated epithelium. An increase in both short circuit current and total conductance was detected when these sugars were used. The Km and Vm values for the increase in short circuit current with glucose exhibited Michaelis-Menten kinetics were 2.8 mM and 69 $\mu A/cm^2$, resp. The values were 7.7 mM and 37 $\mu A/cm^2$, resp., with 3-*O*-methyl-D-glucose, and 47.6 mM and -61.7 $\mu A/cm^2$ with 2-deoxy-D-glucose. It was possible to determine a linear relationship between the two variables and to study the kinetics of the interaction of glucose and 3-*O*-methyl-D-glucose with the sodium sugar carrier by using increasing concentrations of these substances. The increases in short circuit current and total conductance were also verified in isolated epithelium. Phlorizin (5×10^{-6}-10^{-5} M), or the withdrawal of glucose reversed the increase in short circuit current, but the total conductance remained unchanged. Increasing concentrations of 2-deoxy-D-glucose added to the mucosal bathing medium caused a decrease in short circuit current without changing the total conductance, either in preparations of intact ileum or in isolated epithelium.

0056 MECHANICAL PROPERTIES OF TAENIA COLI SMOOTH MUSCLE IN SPONTANEOUS CONTRACTION. (Eng.) Price, J. M.; Patitucci, P.; Fung, Y. C. (Coll. Medicine, Univ. South Florida, Tampa, FL 33612). *Am. J. Physiol.* 233(1):C47-C55; 1977.

To determine the dependence of mechanical properties on the length of the tenia coli smooth muscle, the following mechanical tests were carried out in spontaneously contracting guinea pig tenia coli: isometric step stretch, isotonic step loading, and length-tension. Passive stress-strain tests were also performed. Information on spontaneous contraction was more readily obtained in isometric tests. In length-tension tests, each isometric contraction was analyzed on a phase plane, which is a representation of muscle stress, $\sigma(t)$, versus the rate of change of stress, $\sigma(t)$. The curve in the phase plane provided parameters for describing spontaneous contraction as a function of muscle length, L. The maximum active stress in a cycle of spontaneous contraction, $S(L)_{max}$, and the maximum rate of increasing active stress, $[S(L)]_{max}$, where shown to depend on L in the same way. Both parameters attained a maximum value (upper bound) at the same muscle length, L_{max}. When L was less than L_{max}, the minimum stress in a spontaneous contraction, $\sigma(L)_{min}$ was negligible. If L was greater than L_{max}, $\sigma(L)_{min}$ increased nonlinearly and was equal to the passive stress in an epinephrine-relaxed muscle. The results show that the ratio of $[S(L)]_{max}$ to $S(L)_{max}$ is constant for a physiological range of muscle length. This suggests that the ratio of $[S(L)]_{max}$ to $S(L)_{max}$ may be used as an index of contractility for tenia coli.

0057 PATTERNS OF GASTRIC ELECTRICAL AND MOTOR ACTIVITY IN MINIATURE PIGS. (Eng.) Roze, C.; Couturier, D.; Debray, C. (Hopital Bichat, 170 Bd Ney, 75877 Paris, Cedex 18, France). *Can. J. Physiol. Pharmacol.* 54(5):764-773; 1977.

Electrical and motor gastric patterns were recorded

with chronically implanted electrodes and strain gauge force transducers in fasted and fed miniature Pitman Moore pigs to determine the usefulness of this model for physiological and pharmacological research. The patterns of gastric myoelectrical activity in pigs were generally similar to those observed in dogs, including regular cyclic potential variations in the distal two-thirds of the gastric wall and spike response activity following pacesetter potentials (PP), which was associated with muscle contraction. In fasting animals the frequency and propagation velocity of PP in the distal stomach (<11 cm from the pylorus) ranged from 3.78 ± 0.129 to 4.66 ± 0.130 cycles/min and 4.9 ± 0.18 to 7.8 ± 0.31 mm/sec, resp. There was a significant ($p<0.001$) level of variance in these parameters between different animals studied and between sessions for each animal. The following electrical and motor activity patterns were found to be peculiar to the miniature pigs studied: conduction velocity of the PP was increased only moderately in the antrum compared with a fourfold increase for dogs; tachygastria never occurred in pigs; a slight and progressive increase in PP frequency was observed in response to a standard meal and lasted for 2-4 hr; meal-induced contractions were of large amplitude and proceeded immediately at the maximal contracting force of the stomach; and the periodic reinforcement of spiking that is characterized in dogs by the migrating electrical complexes was not observed. The 40-min period following the administration of a test meal appeared especially suitable for pharmacological or physiological experiments in which inhibitory factors on the stomach are tested.

0058 MEAN TRANSIT TIME OF SOLID TEST MEALS IN GASTRIC EMPTYING. (Eng.) Aeberhard, P.; Steudler, G.; Noelpp, U.; Rosler, H. (Inselspital, CH-3010 Bern, Switzerland). *Digestion* 15(5):361-372; 1977.

Gastric emptying of a technetium-labeled test meal was studied in nine healthy volunteers using a gamma-camera with on-line computer. Mean transit time (MTT) was determined by fitting the time-activity histogram to a gamma-variate, and the half-time of emptying ($T_{\frac{1}{2}}$) was calculated by exponential fitting of the down slope of the time-activity curve. MTT showed a smaller coefficient of variance than $T_{\frac{1}{2}}$. This result suggests that MTT may be a more suitable parameter for definition of normal values and for the separation of normal from pathological patterns of emptying.

0059 THE ROLE OF THE EXTRINSIC ANTRAL NERVES IN THE REGULATION OF GASTRIC EMPTYING. (Eng.) Mroz, C. T.; Kelly, K. A. (Mayo Medical Sch., Rochester, MN). *Surg. Gynecol. Obstet.* 145(3):369-377; 1977.

The hypothesis that extrinsic antral nerves enhance gastric emptying of solids both by initiating and strengthening the bursts of powerful contractions that occur periodically during fasting and by stimulating antral peristalsis and trituration during feeding was tested. In six conscious dogs with chronically implanted gastric and duodenal electrodes, gastric emptying of 40 radiopaque plastic spheres (7 mm diameter, specific gravity 1.6) was assessed fluoroscopically during fasting and after the feeding of boiled beef liver (25 g of 1-cm cubes). Gastric emptying of 400 ml of 154 mM sodium chloride marked with ^{14}C-polyethylene glycol was also measured. The extrinsic antral nerves were then transected, and after recovery, the tests were repeated. Before antral denervation, gastric spheres were emptied predominantly during activity fronts--the 5- to 15-min bursts of action potentials and contractions that sweep aborally from the stomach to the colon every 2 hr during fasting. Feeding abolished activity fronts for about 8 hr, and, therefore, delayed gastric emptying of the spheres. After antral denervation, antral activity fronts were irregular, less frequent, and of shorter duration, and the spheres emptied slower both during fasting and after feeding. In contrast, the emptying of isotonic sodium chloride was unchanged. It is concluded that antral extrinsic nerves enhance gastric emptying of solid spheres but not of isotonic sodium chloride.

0060 PROLONGATION OF GASTRIC EMPTYING BY ORAL PROPANTHELINE. (Eng.) Hurwitz, A.; Robinson, R. G.; Herrin, W. F. (Univ. Kansas Medical Center, Kansas City, KS 66103). *Clin. Pharmacol. Ther.* 22(2):206-210; 1977.

The effect of a frequently used p.o. dose of propantheline bromide (30 mg) on gastric emptying of a radioisotope-labeled test meal was studied. In a prospective, double-blind, randomized crossover design, 13 normal subjects received propantheline or placebo 90 min before taking a 113m-indium-labeled liquid test meal, the volume of which was adjusted to body weight. The disappearance of radioisotope from the area of the stomach was determined by external gamma counting. After placebo, the mean half-emptying time was 68 min, and after propantheline it was 135 min ($p<0.005$). Although salivary flow decreased and pulse rate increased, there were no visual disturbances. This study demonstrates that a single 30-mg dose of propantheline bromide doubles the mean gastric half-emptying time in man.

0061 MYOELECTRICAL AND MECHANICAL ACTIVITY OF STOMACH AND INTESTINE IN HYPOTHYROID DOGS. (Eng.) Kowalewski, K.; Kolodej, A. (Surgical-Medical Res. Inst., Univ. Alberta, Edmonton, Alberta T6G 2N8, Canada). *Am. J. Dig. Dis.* 22(3):235-240; 1977.

Myoelectrical activity was studied in normothyroid and hypothyroid dogs in the resting state, following injection of pentagastrin (6 µg/kg, i.m.) or bethanechol chloride (50 µg/kg, i.m.), or after feeding. Hypothyroidism was produced by total thyroidectomy and chronic treatment with thiouracil. Electrodes were surgically implanted over the serosa of the

stomach, duodenum, jejunum, and ileum and used for repeated recording of myoelectrical activity of gastrointestinal muscles. A strain gauge was implanted over the gastric antrum, and served to record mechanical activity of the stomach. Compared with controls, hypothyroid animals showed decreased frequency of electrical control activity of the stomach (22% decrease, $p<0.01$) and jejunum ($p<0.01$), decreased occurrence of electrical response activity (spike potentials) following stimulation, and decreased mechanical response to the same stimulants, i.e., pentagastrin, bethanechol chloride, and food. The abnormalities in the motor activity of the gastrointestinal muscles in clinical and experimental thyroid deficiency may be due to metabolic and functional changes in the muscle cells.

0062 STIMULATORY (H_1) AND INHIBITORY (H_2) HISTAMINE RECEPTORS IN GALLBLADDER MUSCLE. (Eng.) Waldman, D. B.; Zfass, A. M.; Makhlouf, G. M. (Medical Coll. Virginia, Richmond, VA 23298). *Gastroenterology* 72(5):932-936; 1977.

The nature of histamine receptors in guinea pig gallbladder muscle was examined using specific histamine-receptor agonists and antagonists. Metiamide (10^{-4}-10^{-3} M) augmented the contractile response to histamine, indicating that gallbladder muscle possessed stimulatory H_1 receptors and inhibitory H_2 receptors. The independent inhibitory character of H_2 receptors was confirmed by (a) induction of relaxation with histamine after H_1-receptor blockade and the suppression of this relaxation with metiamide, and (b) induction of relaxation with 4-methyl histamine, and the suppression of this relaxation with metiamide. Blockade of H_2 but not of H_1 receptors augmented the response to the octapeptide of cholecystokinin. The nature of this effect was such that the apparent affinity of the octapeptide for its own receptor was increased. The findings raised the possibility that, in their native unoccupied state, H_2 receptors may modify the response to hormonal agents.

0063 THE SIGNIFICANCE OF IN VITRO EXPERIMENTS FOR THE INVESTIGATION OF GASTROINTESTINAL MOTILITY. (Ger.) Jennewein, H. M.; Weiser, H. F. (Firma Boehringer Sohn, Pharmaforschung Biologie, 6507 Ingelheim am Rhein, W. Germany). *Z. Gastroenterol.* 15(3):161-166; 1977.

0064 RADIOLOGICAL STUDIES OF ESOPHAGEAL AND GASTRIC MOTILITY. (Ger.) Faust, H. (Kantonsspital Basel, CH-4031 Basel, Switzerland). *Z. Gastroenterol.* 15(3):175-184; 1977.

0065 RECORDING OF THE ELECTRICAL ACTIVITY OF THE STOMACH AND INTESTINE. (Pol.) Knapik, Z.; Jagielski, J.; Nowicki, G.; Lubczynska-Kowalska, W.; Cader, J.; Sajewicz, Z. (Z Kliniki Gastroenterologiczna Instytutu Chorob Wewnetrznych ul. Poniatowskiego 2, Wroclaw, Poland). *Pol. Tyg. Lek.* 31(42):1787-1790; 1976.

0066 GASTRIC AND INTESTINAL BIOPOTENTIAL RECORDING IN A CLINIC. (Rus.) Rebrov, V. G. (N. N. Burdenko Principal Military Hosp., USSR). *Klin. Med.* 55(2):24-30; 1977.

0067 CHARACTERISTICS OF THE SKIN ELECTROGASTROGRAPHY AND BALLOON MECHANOGRAPHY OF THE STOMACH MOTOR ACTIVITY. (Rus.) Galperin, Yu. M.; Rebrov, V. G.; Popova, T. S.; Gorin, A. S.; Oparin, V. S. (Sklifossofsky's First Aid Inst., Moscow, USSR). *Fiziol. Zh. SSSR* 62(11):1667-1675; 1976.

0068 THE GASTROINTESTINAL MYOELECTRICAL COMPLEX. (Ger.) Aeberhard, P. (Klinik fur viszerale Chirurgie, Inselspital, CH-3010 Bern, Switzerland). *Z. Gastroenterol.* 15(3):202-208; 1977.

0069 EFFECT OF CORTICOSTERONE, GLUCAGON AND GROWTH HORMONE ON GASTROINTESTINAL PROPULSIVE MOTILITY IN RATS. (Cze.) Visnovsky, P. (Katedra patologickej fyziologie a farmakologie LFUK Partizanska 19, 03753 Martin, Czechoslovakia). *Bratisl. Lek. Listy* 65(4):394-399; 1976.

0070 MOTOR FUNCTION OF THE DUODENUM IN CHILDREN, AS EVIDENCED BY X-RAYS. (Rus.) Filippkin, M. A. (Central Medical Advanced Training Inst., Moscow, USSR). *Vopr. Okhr. Materin. Det.* 21(9):28-31; 1976.

0071 ON THE SPASMOLYTIC ACTION OF 3-BUTOXY-1-PHENOXY-PROPANOL-(2) (FEBUPROL) ON THE BILIARY TRACT. (Ger.) Eisenburger, R. (Klinge Pharma GmbH & Co., Postfach 40 04 20, D-8000 Munchen 40, W. Germany). *Arzneim. Forsch.* 27(II, 7):1429-1433; 1977.

0072 THE EFFECT OF ADRENAL INSUFFICIENCY ON GALLBLADDER MOTILITY. (Rus.) Potapov, A. A.; Potapova, Z. G.; Medvedev, M. A. (Medical Inst., Tomsk, USSR). *Fiziol. Zh. SSSR* 62(11):1691-1697; 1976.

0073 THE INFLUENCE OF VAGOTOMY ON GALLBLADDER MOTILITY. (Rus.) Zhuk, A. M. (Surgical Clinic, S. M. Kirov Military Medical Acad., Leningrad, USSR). *Vestn. Khir.* 118(2):20-24; 1977.

See also, 0007, 0012, 0205, 0309, 0310, 0388, 0441, 0453, 0463.

0074 POTENTIAL AND LIMITATIONS OF ENZYME CYTOCHEMISTRY: STUDIES OF THE INTRACELLULAR DIGESTIVE APPARATUS OF CELLS IN TISSUE CULTURE. (Eng.) Hundgen, M. (Dept. Zoology, Univ. Bonn, Bonn, W. Germany). *Int. Rev. Cytol.* 48:281-321; 1977.

0075 EFFECT OF ELECTRICAL STIMULATION OF THE AUTONOMIC NERVES ON THE LEVELS AND SYNTHESIS OF CYCLIC NUCLEOTIDES IN THE RAT SALIVARY GLANDS: RELATIONSHIP TO ENHANCED GROWTH. (Eng.) Templeton, D.; Butcher, F. R.; Turner, C. J.; Muir, T. C.; Durham, J. P. (Dept. Pharmacology, Univ. Glasgow, Glasgow, Scotland). *J. Cyclic Nucleotide Res.* 3(2):107-118; 1977.

0076 COMPARTMENTATION OF ENZYMES IN THE RABBIT PAROTID SALIVARY GLAND. A STUDY BY ENZYME HISTOCHEMICAL, TISSUE FRACTIONATION AND MORPHOMETRIC TECHNIQUES. (Eng.) Pratten, M. K.; Williams, M. A.; Cope, G. H. (Dept. Human Biology, Univ. Sheffield, Sheffield, England). *Histochem. J.* 9(5):573-598; 1977.

0077 HISTOCHEMISTRY OF 11β-HYDROXYSTEROID DEHYDROGENASE IN RAT SUBMANDIBULAR GLAND. EFFECT OF CORTISOL STIMULATION. (Eng.) Hoyer, P. E.; Moller, M. (Lab. Cyto- and Histochemistry, Univ. Copenhagen, Universitetsparken 1, 2100 Copenhagen, Denmark). *Histochem. J.* 9(5):599-618; 1977.

0078 CHANGES IN THE ATP POOL OF PAROTID AND SUBMAXILLARY GLANDS OF RATS AFTER STIMULATION WITH ISOPROTERENOL. (Eng.) Capps, M. J.; Mooney, C. J.; Itzhaki, S. (Dept. Medical Biochemistry, Univ. Manchester, Stopford Bldg., Manchester M13 9PT, England). *Gen. Pharmacol.* 8(2): 129-131; 1977.

See also, 0001, 0005, 0006, 0242, 0527, 0536.

Stomach

0079 GLYCOPROTEIN SYNTHESIS IN GASTRIC EPITHELIAL CELLS OF THE RAT. PROPERTIES OF MICROSOMAL GLYCOPROTEIN GLYCOSYLTRANSFERASES. (Eng.) Strous, G. J. A. M.; Kramer, M. F. (Dept. Histology Cell Biology, Univ. Utrecht, Nic. Beetsstraat 22, Utrecht, Netherlands). *Biochim. Biophys. Acta* 451(1):201-211; 1976.

The mechanism of the biosynthesis of the epithelial glycoproteins that line the inner surface of the mammalian stomach was investigated in tissue preparations from the Wistar rat. An assay method was devised for determining microsomal glycoprotein galactosyl- and fucosyltransferase in gastric scrapings, using both exogenous and endogenous glycoprotein. Subcellular fractionation of the homogenate yielded microsomal fractions that were high in the glycosyltransferases, with the probable location of fucosyltransferase on the rough endoplasmic reticulum and the galactosyltransferases on the Golgi apparatus. The amount of galactosyltransferase in the total cell homogenate was increased when studied after rats were starved for 48 hr and then permitted access to food for 3 hr (0.51 ± 0.034 nmol/min/μg DNA versus 0.65 ± 0.052 nmol/min/μg DNA, $p<0.01$). The endogenous acceptor molecule was identified as a glycoprotein.

0080 MOLECULAR FORMS OF GASTRIN IN THE ANTRAL MUCOSA. PLASMA AND GASTRIC JUICE DURING VAGAL STIMULATION OF ANESTHETIZED CATS. (Eng.) Unvas-Wallensten, K.; Rehfeld, J. F. (Karolinska Institutet, Stockholm, Sweden). *Acta Physiol. Scand.* 98(2):217-226; 1976.

Antral mucosa, blood, and gastric juice samples collected from anesthetized cats during electrical vagal stimulation were subjected to gel filtration to characterize the different molecular forms of gastrin. The gastrin concentration in the antral mucosa ranged from 2.82 to 3.60 μg/g mucosa, and the predominant gastrin form in antral mucosa was component III (gastrin-17), which accounted for about 94% of the total immunoreactivity. The antrum

also contained 5% component II (gastrin-34) and 1% component I gastrin as well as trace amounts of component IV (gastrin-14). The gastrin concentration in the gastroduodenal vein (mainly draining the antrum) at 30-60 min after the start of vagal stimulation was as high as 12,400 pg/ml. Immediately after vagal stimulation, component III gastrin appeared in the gastric venous effluent followed within a few minutes by component IV gastrin. Component I or II gastrin was not detectable in plasma samples. It is suggested that component III gastrin is released from the antral mucosa and is then rapidly metabolized to component IV gastrin, possibly to a significant extent in the fundic region of the stomach. Gastrin levels of approximately 45,000 pg/ml were found in some gastric juice samples that contained large amounts of component III. Only very small amounts of immunoreactive degradation products were present in gastric juice, indicating that cat gastrin is relatively resistant to peptic degradation and acid hydrolysis.

0081 CHARACTERIZATION OF AN ADENYLATE CYCLASE SYSTEM SENSITIVE TO HISTAMINE H_2-RECEPTOR EXCITATION IN CELLS FROM DOG GASTRIC MUCOSA. (Eng.) Scholes, P.; Cooper, A.; Jones, D.; Major, J.; Walters, M.; Wilde, C. (Imperial Chemical Industries Ltd., Pharmaceuticals Div., Mereside, Alderley Park, Macclesfield, Cheshire SK 10 4TG, England). *Agents Actions* 6(6):677-682; 1976.

The effect of histamine on the cyclic AMP level was investigated in canine gastric mucosal cells. Cyclic AMP in these cells was elevated by histamine and 4-methyl histamine, but 2-methyl histamine was only a weak agonist. The effects on cyclic AMP levels were inhibited competitively by metiamide and burimamide, which gave apparent K_B values of 3.5×10^{-7} M and 2.3×10^{-6} M, resp. These values are similar to those reported for other histamine H_2-receptor systems. The H_1-receptor antagonists, mepyramine and chlorpheniramine, had no inhibitory effect on the histamine-induced elevation of cyclic AMP; promethazine inhibited the system, but not by a competitive mechanism. It is concluded that the histamine-stimulated adenylate cyclase system is probably located in the parietal cell component.

0082 EFFECT OF ATROPINE AND METHIAMIDE ON VAGALLY INDUCED GASTRIC ACID SECRETION AND GASTRIN RELEASE IN ANESTHETIZED CATS. (Eng.) Uvnas-Wallensten, K.; Andersson, H. (Karolinska Institutet, Stockholm, Sweden). *Acta Physiol. Scand.* 99(4):496-502; 1977.

The response of vagally-stimulated gastric acid secretion and gastrin release to atropine and methiamide was investigated in anesthetized cats. The vagal nerves, cut in the cervical region, were electrically stimulated (7 V, 2-5 msec, 2-10 Hz) for 1-3 hr to study gastric acid secretion or 5 or 10 min to study gastrin release. Atropine (0.1-0.3 mg/kg) or methiamide (4, 10, and 20 mg/kg) was administered 30-60 min after the beginning of electrical stimulation, and mean acid output was calculated before (last 15 min) and after (first two 15-min periods) the drug administration. To study gastrin release, atropine (0.1-2 mg/kg) or methiamide (20 mg/kg) were administered 15 min after the first and 10 min prior to the second electrical stimulation. Atropine reduced vagally stimulated gastric acid secretion approximately 40 and 30% of control values in the first and second 15-min periods following drug administration, resp. Methiamide reduced gastric acid secretion to about 30 and 10% of control values in the first two 15-min periods after drug administration. Gastrin release was not reduced by either drug. Possible explanations for the insensitivity of vagally induced gastrin release to atropine are discussed. It is suggested that the insensitivity of the neuroeffector junctions at the antral G cells to atropine and methiamide may be due to the presence of noncholinergic fibers stimulating the antral G cells or to nonmuscarinic cholinergic receptors.

0083 EFFECT OF HISTAMINE H_2 ANTAGONISM BY METIAMIDE ON THE RESPONSE OF THE CANINE GASTRIC MUCOSA TO ACID AND BILE SALT. (Eng.) Gurll, N. J.; Zinner, M. J.; Callahan, W. (Walter Reed Army Inst. Res., Washington, DC). *Gastroenterology* 73(2):255-259; 1977.

To clarify the effect of histamine H_2-receptor antagonists on gastric mucosa damaged by acid or acid plus sodium taurocholate, the effects of i.v. metiamide infusion on three characteristics of the gastric mucosa (the ionic fluxes of Na^+ and H^+, transmural potential difference [PD], and protein loss into the gastric lumen) were determined in five awake mongrel dogs. Denervated Heidenhain (fundic) pouches were prepared in the dogs, and antrectomy was performed followed by gastroduodenostomy. Experiments began 3 weeks later, and each dog was tested once a week for 2 months. The pouch lumen was perfused with 70 ml of either 80 mM HCl plus 80 mM NaCl (ATS) or ATS plus 20 mM sodium taurocholate (BS) for 3 hr at 37 C. Metiamide or 0.9% NaCl was infused i.v. at 10 μmol/kg/hr, beginning 30 min prior to perfusion of the pouch. Each dog was tested twice with each of the four possible combinations of agents. Exposure to ATS produced increases in the Na^+, Cl^-, and K^+ outputs, as well as in the luminal volume. During exposure to ATS, metiamide increased the mean H^+ clearance rate from -0.5 ± 0.5 to -1.9 ± 0.5 ml/30 min ($p<0.05$), but did not affect the PD, ion fluxes, or luminal volume. Exposure to BS increased the net Na^+ flux threefold and the K^+ gain by fourfold, in addition to increasing the volume and H^+ clearance rate. Metiamide infusion did not significantly alter the total mean H^+ clearance rate or ion fluxes during exposure to BS. The mean hourly net H^+ loss was significantly increased ($p<0.05$) by metiamide during the first 2 hr of exposure to BS even though H^+ concentration and PD were not affected. In addition, Cl^- gain was much lower than control values (BS alone) during the second and third hours, suggesting that acid secretion was reduced; this finding may also explain the increase in H^+ loss.

Metiamide infusion reduced the protein loss during exposure to BS in three of five dogs from >50 mg/3 hr to <20 mg/3 hr. These observations suggest that histamine may mediate gastric mucosal damage, particularly that resulting from exposure to BS.

0084 METIAMIDE AND STIMULATED ACID SECRETION FROM THE ISOLATED NON-DISTENDED AND DISTENDED MOUSE STOMACH. (Eng.) Wan, B. Y. C. (Univ. Coll. London, Gower St., London WC1E 6BT, England). *J. Physiol. (Lond.)* 266(2):327-346; 1977.

The action of metiamide on the acid secretory response to various gastric stimuli in the perfused, isolated, distended, or nondistended mouse stomach was investigated. The distended stomach gave a marked and dose-related acid secretory response to histamine (HA, 10^{-6}-10^{-3} M), pentagastrin (PG, 10^{-8}-10^{-5} M), acetylcholine (5 x 10^{-5}-10^{-5} M), eserine (10^{-5}-10^{-3} M), or dibutyryl cyclic AMP (dbcAMP, 5 x 10^{-5}-10^{-3} M). In the nondistended stomach, dbcAMP regularly stimulated acid secretion in a dose-dependent manner; HA, PG, or acetylcholine did not always stimulate acid secretion. HA or PG, but not acetylcholine, always caused significant stimulation of acid secretion from the nondistended stomach in the presence of a phosphodiesterase inhibitor such as caffeine, theophylline, or the I.C.I. compound, 63197. At 10^{-4} M, these phosphodiesterase inhibitors markedly potentiated the stimulatory effect of HA or PG on acid secretion, and the order of effectiveness was 63197>theophylline>caffeine. 63197 Potentiated HA- or PG-stimulated acid secretion in the distended stomach markedly. Metiamide (5 x 10^{-5}-10^{-4} M) did not antagonize stimulation of acid secretion by dbcAMP in the nondistended or distended stomach. In the distended stomach, metiamide (5 x 10^{-5} M) significantly inhibited HA-stimulated acid secretion with a linear and parallel displacement of the HA dose-response curve to the right. Although at this concentration metiamide did not depress the maximal acid secretory response to HA, it markedly reduced the maximal acid secretory response attainable with PG. In the distended stomach, metiamide (5 x 10^{-5} M) did not inhibit acetylcholine-induced acid secretion significantly. Atropine (5 x 10^{-6} M) abolished the stimulatory effect of acetylcholine; it also inhibited PG-stimulated acid secretion, but it had little effect on acid secretion induced by HA. The results indicate that metiamide inhibits HA-induced acid secretion by competitive antagonism of the HA H_2-receptor, but its inhibitory effect on PG-induced acid secretion seems to be of a noncompetitive nature. The failure of metiamide to inhibit acid secretion induced by dbcAMP suggests that cAMP might regulate gastric acid secretion at a site beyond the H_2-receptor activation. The present results support the hypothesis that cAMP may be involved in HA-induced acid secretion in the isolated mouse stomach.

0085 EFFECT OF HISTAMINE ON TECHNETIUM-99m EXCRETION BY GASTRIC MUCOSA. (Eng.) Wine, C. R.; Nahrwold, D. L.; Rose, R. C.; Miller, K. L. (Milton S. Hershey Medical Center, Hershey, PA 17033). *Surgery* 80(5):591-594; 1976.

The effects of histamine on ^{99m}technetium (Tc) pertechnetate (TcP) excretion by denervated antral and oxyntic gland mucosa were studied in four dogs prepared with a separated denervated antral pouch and a Heidenhain pouch and fasted for 18 hr prior to secretory studies. After an infusion of 0.15 M sodium chloride for two 15-min periods, histamine dihydrochloride (0.01, 0.02, or 0.16 mg/kg/hr) was constantly infused for twelve 15-min periods. After histamine had been infused for four 15-min periods, the dogs received a single rapid i.v. injection of TcP (0.9-1.6 mCi). All histamine doses caused a significant increase in Heidenhain pouch acid output that plateaued within 1 hr after the start of histamine infusion. Acid output was not affected by the TcP injection. Heidenhain pouch TcP output increased with increasing histamine doses. Peak TcP output (about 13 µCi/15 min) occurred at 30 min after a 0.16 mg/kg/hr histamine treatment and gradually declined thereafter. Peak TcP output (about 7 µCi/15 min) with a 0.02 mg/kg/hr histamine dose also occurred at 30 min after the start of histamine infusion, but the output remained at about the same level thereafter. With a histamine dose of 0.01 mg/kg/hr, there was a gradual increase in TcP output that reached about 6 µCi/15 after 2 hr of histamine infusion. When the mean total 2-hr TcP output was plotted against the mean 2-hr volume or acid output of gastric juice from Heidenhain pouches, a linear relationship was found in each case. Antral pouch secretion studies revealed no significant differences in pH, antral secretion volume, or antral pouch TcP output at different histamine doses. The maximum mean 2-hr TcP output from antral pouches (7.9 ± 0.9 µCi/2 hr) obtained with the highest histamine dose was less than one-third of the mean 2-hr output from Heidenhain pouches (26.6 µCi) obtained with the lowest histamine dose. The results suggest the possibility that TcP scanning accuracy in Meckel's diverticulum might be improved by the administration of histamine prior to TcP injection.

0086 EFFECT OF CALCITONIN ON GASTRIC SECRETION. (Eng.) Yonaga, T.; Nakamura, K.; Matsuura, K. (Teikyo Univ. Sch. Medicine, Kaga 2-11-1, Itabashi-ku, Tokyo 173, Japan). *Proc. Jpn. Acad.* 52(9):532-535; 1976.

The mechanism of action of calcitonin (CT) on gastric secretion in rats with chronic gastric fistulas was investigated. The relation between CT action and serum calcium level was also studied in rats given an infusion of histamine (1 mg/hr) and calcium gluconate (2.5 mg/hr). Volume and acid and pepsin concentrations were measured in samples of gastric secretion collected every 30 min from rats injected s.c. with synthetic salmon CT (1 or 5 IU) dissolved in 1% acetic acid. Prior to the CT injection, rats had received saline alone (1.6 ml/hr infused through tail vein), saline infusions containing a stimulant of gastric secretion (histamine, 1 mg/hr; tetragastrin, 0.8 µg/hr; or bethanechol chloride,

20 µg/hr), or 2-deoxy-D-glucose (20 mg/rat), which is thought to stimulate the vagus nerve. Results revealed that CT completely inhibited stimulated and nonstimulated gastric secretion, although the degree of inhibition varied under different conditions. Gastric acid and pepsin secretions stimulated with histamine and calcium gluconate infusions were also inhibited by s.c. CT administration, indicating that the effect of CT on gastric secretion is independent of serum calcium levels.

0087 RELATIONSHIP OF THE GASTRIN RESPONSE TO THE AMOUNT OF FOOD INGESTED IN NORMAL SUBJECTS. (Eng.) Woussen-Colle, M. C.; Willems, G.; De Graef, J. (Hospital Saint-Pierre, Brussels, Belgium). *Digestion* 15(4):322-328; 1977.

The effect of the quantity of food ingested on the gastrin response was studied in eight normal subjects who received three different amounts of the same meal. The subjects received a meal that included 150 g of steak on one occasion, and on two other occasions one-half or twice this amount, resp. The peak gastrin response was similar for the three meals, but the integrated gastrin response was larger with the largest meals. A comparison of the response to the two largest meals indicated that in a given subject, the gastrin response is similar when tested on different occasions. A good correlation was found between individual fasting concentrations of gastrin and peak gastrin levels after feeding but not between basal gastrin and the integrated gastrin response. This finding could indicate that in normal subjects, the first two parameters are primarily related to the same control mechanisms (possibly the G cell mass), while the integrated gastrin response reflects the effectiveness of the feedback mechanisms controlling gastrin release.

0088 RESERPINE, CATECHOLAMINES AND VAGOTOMY IN THE RAT GASTRIC MUCOSA. AN ELECTRON MICROSCOPIC STUDY. (Eng.) Nyhus, L. M.; Kalahanis, N. G.; Das Gupta, T. K. (Abraham Lincoln Sch. Medicine, Chicago, IL). *Chir. Gastroenterol.* 10(4): 359-365; 1976.

The effects of reserpine on the enterochromaffin-like (ECL) cells of the rat fundic mucosa were examined. Fundic mucosa from control and reserpine (2.5 mg/kg x 2 days, i.p.)-treated animals was examined ultrastructurally. Reserpine caused an increase in the synthesis and release of ECL cell catecholamines. The increased local release of catecholamines may account for the ulcerogenic properties of reserpine. Previous studies have shown that vagotomy depletes the ECL cells of their granular content with no simultaneous increase in its synthesis; this effect may be one of the mechanisms whereby vagotomy prevents ulcer formation in reserpine-treated rats.

0089 EFFECT OF LYSINE-ACETYLSALICYLATE ON SERUM GASTRIN LEVELS. (Eng.) Estler, C. J.; Mitznegg, P.; Domschke, W.; Demling, L. (Dept. Pharmacology, Univ. Erlangen-Nurnberg, Universitatssr. 22, D-8520 Erlangen, W. Germany). *Acta Hepatogastroenterol.* 24(1):52-54; 1977.

The effect of lysine-acetylsalicylate (LAS) on serum gastrin levels was investigated in mice. Intragastrically administered LAS did not alter serum gastrin values, while a very high dose of LAS (450 µg/g) caused a short-lived, significant increase in serum gastrin (74.3 ± 17.1 versus 36.9 ± 7.6 pg/ml in controls, $p \leq 0.05$) when administered i.v. These results are consistent with the view that salicylates do not owe their potential ulcerogenic properties to stimulation of the gastrin-gastric secretion mechanism.

0090 PARIETAL CELLS OF THE STOMACH TRAP SALICYLATES DURING ABSORPTION. (Eng.) Brune, K.; Schweitzer, A.; Eckert, H. (Biocenter, Klingelbergstrasse 70, CH-4056, Basel, Switzerland). *Biochem. Pharmacol.* 26(18):1735-1740; 1977.

Autoradiographic methods were used to study the uptake of salicylates by the parietal cells in the stomach of rats. Whole-body autoradiographs taken 45 min after the administration of the ^{14}C-salicylic acid (10 µCi) showed activity in the kidneys and stomach wall but not in the stomach lumen or gut. Additional studies of thin slices of the stomach wall removed 1, 5, and 15 min after ^{3}H-salicylic acid administration indicated very rapid diffusion of the drug across the gastric wall in the glandular stomach. A method was devised for the rapid fixation of both the tissue and drug for autoradiography (immediate deep freezing of stomach wall in liquid propane, cutting in a cryostat at -25 C, mounting on emulsion and exposure at the same temperature). High drug concentrations were observed throughout the glandular stomach, but particularly in the parietal cells within 1 min of drug administration, indicating selective trapping of salicylate molecules by parietal cells. It is hypothesized that decay of the parietal cells is initiated by the salicylate immediately, and ulcers may be the ultimate outcome of this process. Results support the assumption that functioning parietal cells are essential for the development of ulcers due to salicylate medication, which would explain why achlorhydric patients rarely experience gastric side-effects from aspirin.

0091 THE EFFECT OF SEROTONIN ON PEPSIN INHIBITION BY DUODENAL FAT. (Eng.) Wazna, M. F.; Stein, T.; Wise, L. (Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 11040). *Ann. Surg.* 186(2):130-135; 1977.

The role of serotonin in pepsin secretion was studied in dogs. Twelve female mongrel dogs were prepared with a Heidenhain pouch and gastric and duodenal cannulae. The purpose of the gastric cannulae was to divert stomach acid from the duodenum to prevent inhibition of gastric secretion during the experiment. Pretreatment with reserpine (0.1 mg/kg, i.m.)

abolished the stimulatory effect of histamine (0.04 mg/kg/hr) on pepsin secretion, while pretreatment by 1-methyl-d-lysergic acid butanolamide (2 mg/hr, i.v.) increased the pepsin inhibition resulting from the intraduodenal infusion of corn oil emulsion. 5-Hydroxytryptophan (20 mg/kg, i.v.) injected simultaneously with the intraduodenal corn oil caused marked increase in pepsin secretion. Since these effects were observed in the denervated Heidenhain pouch, it is suggested that serotonin acts directly as a hormone on gastric chief cells.

0092 PRINCIPAL REGULATORY FACTORS OF GASTRIC ACID SECRETION. (Ita.) Crema, A.; Frigo, G. M. (Istituto di Farmacologia Medica dell'Universita, Piazza Botta 10, 27100 Pavia, Italy). *Minerva Med.* 68(5):319-329; 1977.

0093 ENTERAL OXYGENATION AND ACID FORMING GASTRIC FUNCTION. (Rus.) Anisimov, A. V.; Erdeli, V. V. (Gorkii Medical Inst., Gorkii, USSR). *Vrach. Delo* (2):105-106; 1977.

0094 HYDROCHLORIC ACID SECRETION BY THE GASTRIC MUCOSA OF THE RAT FETUS. (Fre.) Geloso, J. P.; Garzon, B.; Vassout, P. (Laboratoire de Physiologie animale, Universite Paris 7, Bat. L, 2, place Jussieu, 75005 Paris, France). *C.R. Acad. Sci.* [*D*] *(Paris)* 285(10):1209-1211; 1976.

0095 INHIBITORY EFFECT OF AN H_2 RECEPTOR ANTAGONIST OF HISTAMINE (CIMETIDINE) ON GASTRIC SECRETION [Abstract]. (Fre.) Toulet, J.; Amrani, N.; Parmentier, T.; Bose, F.; Etcheverry, M.; Kerdiles, R.; Rousset, N. (Hopital International, Universite de Paris, 42, boulevard Jourdan, 75014 Paris, France). *Gastroenterol. Clin. Biol.* 1(5): 489-490; 1977.

0096 PENTAGASTRIN, HISTAMINE AND ASCORBIC ACID IN RAT GASTRIC MUCOSA. (Fre.) Gairard, A.; Pestis, J. (Laboratoires de Physiologie, UER des Sciences Pharmaceutiques ULP, 3 rue de l'Argonne, F-67083 Strasbourg Cedex, France). *Experientia* 33(4):457-458; 1977.

0097 INHIBITING ACTION OF TRITHIOZINE (ISF 2001) AND PROPANTHELINE ON HUMAN GASTRIC SECRETION. DOUBLE-BLIND CROSS-OVER STUDY. (Ita.) Bianchi Porro, G.; Ferrara, A.; Petrillo, M.; Abbondati, G. ("L. Sacco" Hosp., Milan, Italy). *Minerva Gastroenterol.* 22(2):149-152; 1976.

0098 INVESTIGATION OF THE THEORETICAL AND PRACTICAL PROBLEMS POSED BY RECENT KNOWLEDGE OF GASTRIN. (Fre.) Bonfils, S.; Bernades, P. (Hopital Bichat, 170, boulevard Ney, F 75877, Paris Cedex 18, France). *Nouv. Presse Med.* 5(35):2317-2321; 1976.

0099 THE EFFECT OF BLOCKING THE CHOLINOREACTIVE SYSTEMS ON THE APPEARANCE OF "STRESS" AND "STEROID" ULCERS OF THE STOMACH. (Rus.) Berger, E. N.; Bondarenko, Yu. I.; Rosolovsky, A. P. (Ternopol Medical Inst., Ternopol, USSR). *Patol. Fiziol. Eksp. Ter.* (1):29-32; 1976.

0100 PHARMACOLOGICAL STUDIES ON THE EFFECTS OF NEW PIPERAZIN DERIVATIVES AND SELECTED DRUGS ON EXPERIMENTAL GASTRIC ULCER. (Pol.) Malecki, I. (Instytut Przemyslu Farmaceutycznego, ul. Rydgiera 8, 01-793 Warsaw, Poland). *Pol. Tyg. Lek.* 31(42):1811-1813; 1976.

0101 INFLUENCE OF PYLORIC STENOSIS ON THE GENESIS OF STRESS ULCER IN THE RAT. (Ger.) Schramm, H.; Rupprecht, H.; Schafer, F. (Chirurgische Universitatsklinik, Bachstrasse 18, DDR-69 Jena, E. Germany). *Z. Exp. Chir.* 10(2):75-78; 1977.

0102 INFLUENCE OF PROSTAGLANDIN E_2 ON THE ACID-FORMING FUNCTION OF THE STOMACH AND ON ULCER FORMATION. (Rus.) Kochina, E. N.; Tsvetkova, L. I.; Grebeneva, L. S.; Polozhenkova, L. A.; Grebenev, A. L. (I. M. Sechenov First Moscow Medical Inst., Moscow, USSR). *Patol. Fiziol. Eksp. Ter.* (3):60-62; 1977.

0103 GASTRIC SECRETION IN RATS WITH PYLORIC LIGATION PERFORMED ACCORDING TO EITHER SHAY'S OR DAI'S METHOD. (Spa.) Cannata, B.; Figueroa, E.; Aguero, E.; Peralta, H.; Suarez, J. R. E. (Facultad de Ciencias Medicas, Universidad Nacional de Cuyo, Venezuela). *Acta Gastroenterol. Latinoam.* 7(1):43-46; 1977.

0104 ROLE OF THE NERVOUS SYSTEM AS A POSSIBLE TRIGGER MECHANISM IN THE PATHOGENESIS OF EXPERIMENTAL GASTRIC ULCERS. (Rus.) Shekhtman, V. M. (Orenburg Medical Inst., Orenburg, USSR). *Patol. Fiziol. Eksp. Ter.* (4):45-48; 1977.

0105 HORMONAL AND NERVOUS CONTROL OF ELECTRICAL AND MECHANICAL FUNCTIONS OF ISOLATED STOMACH. (Pol.) Zajac, S.; Kowalewski, K. (Acad. Medicine, Warsaw, Poland). *Pol. Przegl. Chir.* 49(7):683-690; 1977.

0106 DIGESTIVE PROCESS OF POTATO STARCH GRANULES IN RAT ALIMENTARY TRACT. (Jpn.) Tada, H.; Kawai, F. (Faculty Nutrition, Koshien Univ., Takarazuka, Hyogo, Japan). *J. Jpn. Soc. Food Nutr.* 30(1):15-21; 1977.

See also, 0124, 0217, 0231, 0251, 0259, 0262, 0265, 0267, 0268, 0269, 0281, 0369, 0374, 0377, 0380, 0350, 0362.

0107 PANCREATIC ACINAR CELLS: LOCALIZATION OF ACETYLCHOLINE RECEPTORS AND THE IMPORTANCE OF CHLORIDE AND CALCIUM FOR ACETYLCHOLINE-EVOKED DEPOLARIZATION. (Eng.) Iwatsuki, N.; Petersen, O. H. (Dept. Physiology, Univ. Dundee, Dundee DD1 4HN, Scotland). *J. Physiol. (Lond.)* 269(3):723-733; 1977.

Acetylcholine (ACh) receptors were localized in pancreatic acinar cells, and the importance of chloride and calcium for ACh-evoked depolarization was studied. Intracellular microelectrode recordings of acinar cell membrane potential and resistance were made from the mouse pancreas superfused *in vitro*. The acinar cells were stimulated by microiontophoretic ACh application from an extracellular AChCl-filled microelectrode. Passing short-lasting ejecting current pulses through the AChCl-electrode caused acinar cell depolarization when the electrode was in an extracellular position not far (<50 μm) from an acinus impaled by a KCl microelectrode. After the insertion of the AChCl electrode into a neighboring acinar cell, electrically coupled to the acinar cell already impaled by the KCl-electrode, ejecting ACh current pulses only affected the membrane potential in a direct electrical manner, whereas there was no sign of an effect of ACh on the membrane potential. Replacing extracellular chloride by sulfate caused a marked increase in the amplitude of the ACh-evoked depolarization. If the membrane potential was recorded with a KCl electrode, ACh continued to evoke very large depolarizations even after more than 1 hr exposure to Cl-free solution. If the membrane potential was recorded with a K-citrate electrode, the effect of Cl-removal was only transient. Removal of Na^+ during exposure to Cl-free solution reduced the amplitude of the ACh-evoked depolarization. Readmission of Cl after more than 1 hr of Cl deprivation caused an immediate reversal of the ACh effect into a hyperpolarization. Removal of extracellular Ca^{2+} caused a marked reduction in the amplitude of small depolarizations evoked by just suprathreshold doses of ACh, whereas there was very little effect on larger depolarizations evoked by maximal or supramaximal ACh ejections. The effect of Ca removal was fully reversible. The addition of Mn after Ca-deprivation was as efficient as Ca in restoring normal electrophysiological responses to small doses of ACh. The acinar cell membrane seems only to be responsive to ACh added to the extracellular side, and ACh probably causes an increase in membrane Cl permeability in addition to the previously described effects on Na and K permeability. Ca may be important in determining ACh receptor sensitivity.

0108 PANCREATIC ACINAR CELLS: THE ACETYLCHOLINE EQUILIBRIUM POTENTIAL AND ITS IONIC DEPENDENCY. (Eng.) Iwatsuki, N.; Petersen, O. H. (Dept. Physiology, Univ. Dundee, Dundee DD1 4HN, Scotland). *J. Physiol. (Lond.)* 269(3):735-751; 1977.

To gain insight into the ionic permeabilities of the pathways opened up by acetylcholine (ACh) in the pancreatic acinar cell membrane, the ACh equilibrium potential (E_{ACh}) was determined, and the effects of changes in K, Na, and Cl concentrations were investigated. Two glass microelectrodes were inserted into neighboring cells from rat or mouse pancreatic segments, superfused *in vitro*. The tip of a third glass microelectrode, filled with 2 M AChCl, was placed just outside the acinus. Membrane potential and resistance, and changes in these parameters in response to short pulses of ACh stimulation, were recorded. The resting current-voltage relationship, obtained by injecting 100 msec depolarizing or hyperpolarizing current pulses through one of the intracellular microelectrodes and recording the membrane potential with the other intracellular electrode, was linear within the range -5 to -60 mV. When the acinar cell membrane was hyperpolarized, the amplitude of ACh-evoked depolarization was increased, while ACh-evoked depolarization was reduced when the membrane potential was reduced by depolarizing current, and finally changed into a hyperpolarization at very low membrane potentials. In each acinus investigated (rat and mouse), there was a linear relationship between amplitude of ACh-evoked potential change (ΔV) and resting membrane potential. During superfusion with control solution, the value of the membrane potential at which ACh did not evoke a potential change was about -15 mV in the mouse and about -20 mV in the rat. During superfusion with a chloride-free sulphate-containing solution (steady state), a linear relationship between ΔV and resting membrane potential was again found, but E_{ACh} (mouse) was about +10 mV. Replacing extracellular chloride by sulfate caused an immediate change in E_{ACh} in the positive direction. Readmission of chloride, after a long period of chloride ion deprivation, caused an immediate sharp change in E_{ACh} in the negative direction. Replacing extracellular sodium by Tris caused an immediate transient negative change in E_{ACh}. In contrast, removing extracellular calcium changed E_{ACh} in a positive direction. Augmenting extracellular potassium concentration to 40 mM caused a change in E_{ACh} in the positive direction. At a membrane potential (V) equal to E_{ACh}, the sum of ionic currents evoked by the action of ACh is zero. ACh increased membrane Na, K, and Cl permeability. The approximate relative ion permeabilities of the pathways opened up by ACh are $P_{Na}/P_K=2.5$ and $P_{Cl}/P_K=5$. At $V=E_{ACh}$, the approximate relative sizes of the ACh-evoked currents are $I_{Na}/I_K=2.6$ and $I_{Cl}/I_K=1.6$. ACh, therefore, causes influx of Na and Cl and a small efflux of K.

0109 PARALLEL SECRETION OF DIGESTIVE ENZYMES BY THE IN VITRO RABBIT PANCREAS. (Eng.) Steer, M. L.; Glazer, G. (Beth Israel Hosp., Boston, MA 02215). *Am. J. Physiol.* 231(6):1860-1865; 1976.

The total output of proteins and the secretion of the enzymes, α-amylase, trypsinogen, and chymotrypsinogen by the rabbit pancreas stimulated *in vitro* by methacholine, cholecystokinin-pancreozymin (CCK-PZ), or Sincalide (C-terminal octapeptide of CCK-PZ) was studied. The entire pancreas with an attached loop of duodenum and segment of colon was excised *en bloc* from New Zealand white rabbits and mounted in an incubation chamber. Pancreatic juice samples were collected for 1-hr periods prior to and after addition of the stimulants to the incubation medium. The final concentration of methacholine added was 0.8 mg/100 ml, that of CCK-PZ was 4 Ivy dog U/100 ml, and that of Sincalide was 12 Ivy dog U/100 ml. The protein concentration was significantly increased ($p<0.01$ and $p<0.005$) in all juice samples collected after the addition of stimulants. When the ratio of each enzyme to the others for each animal and for each collection period was measured, only paralleled discharge of the three enzymes was observed. The results are consistent with the mass-transport theory of enzyme synthesis and secretion by the exocrine pancreas.

0110 EFFECT OF GLUCAGON ON THE SECRETORY PROCESS IN THE RAT EXOCRINE PANCREAS. (Eng.) Adler, G. (Dept. Cell Biology and Cell Pathology, Philipps-Univ. Marburg, Robert-Koch-Str. 6, D-3550 Marburg, W. Germany). *Cell Tissue Res.* 182(2): 193-204; 1977.

The effect of glucagon on the enzymes, ultrastructure, and secretion of the pancreas was studied in rats. Glucagon (10-80 µg/hr) was infused i.v. into conscious rats for up to 24 hr. The effect on the secretory process was studied *in vitro* using isolated pancreatic lobules. A pronounced inhibition of the rate of protein synthesis and discharge of stored and newly synthesized proteins, combined with increased enzyme content, were observed after 30 min of infusion. At a glucagon infusion of 50 µg/hr, amylase, lipase, and chymotrypsin levels increased approximately 30% over the control levels. This effect was absent after longer infusion periods of up to 6 hr. After 12- to 24-hr infusions, a marked degranulation and decrease in enzyme content was observed. While the rate of protein synthesis was not significantly enhanced, both the basal and stimulated discharge of enzymes from the pancreas were increased. The results suggest a biphasic response of the pancreas to prolonged glucagon infusion.

0111 Ca^{++} FLUXES IN ISOLATED CELLS OF RAT PANCREAS. EFFECT OF SECRETAGOGUES AND DIFFERENT Ca^{++} CONCENTRATIONS. (Eng.) Kondo, S.; Schulz, I. (Max-Planck-Institut fur Biophysik, 6 Frankfurt [Main], W. Germany). *J. Membr. Biol.* 29 (1/2):185-203; 1976.

The effects of secretagogues and of different calcium concentrations on ^{45}Ca influx and efflux were studied in isolated rat pancreatic cells to characterize the mechanism of Ca^{2+} transport. The secretagogues pancreozymin, carbamylcholine, gastrin I, the octapeptide of pancreozymin, and cerulein, as well as the Ca^{2+}-ionophore A 23187, stimulated ^{45}Ca efflux from isolated cells. The nonsecretagogic hormones isoproterenol, secretin, as well as dibutyryl cyclic AMP and dibutyryl cyclic guanosine 3',5'-monophosphate, had no effect on ^{45}Ca efflux. Atropine blocked the stimulatory effect of carbamylcholine on ^{45}Ca efflux completely, but not that of pancreozymin. A graphical analysis of the Ca^{2+} efflux curves revealed at least three phases: a first phase, probably derived from Ca^{2+} bound to the plasma membrane; a second phase, possibly representing Ca^{2+} efflux from cytosol of the cells; and a third phase, probably from mitochondria or other cellular particles. The Ca^{2+} efflux of all phases was stimulated by pancreozymin and carbamylcholine. Ca^{2+} efflux was not significantly affected by the presence or absence of Ca^{2+} in the incubation medium. Metabolic inhibitors of ATP production, Antimycin A and dinitrophenol, which inhibit Ca^{2+} uptake into mitochondria, stimulated Ca^{2+} efflux from the isolated cells, but inhibited the slow phase of Ca^{2+} influx, indicating the role of mitochondria as an intercellular Ca^{2+} compartment. Measurements of the $^{45}Ca^{2+}$ influx at different Ca^{2+} concentrations in the medium revealed saturation type kinetics, which are compatible with a carrier or channel model. The hormones mentioned above stimulated the rate of Ca^{2+} translocation. The data suggest that secretagogues of pancreatic enzyme secretion act by increasing the rate of Ca^{2+} transport, most likely at the level of the cell membrane, and that Ca^{2+} exchange diffusion does not contribute to the $^{45}Ca^{2+}$ fluxes.

0112 STIMULUS-SECRETION COUPLING IN PANCREATIC ACINAR CELLS: INFLUENCES OF EXTERNAL SODIUM AND CALCIUM ON RESPONSES TO CHOLECYSTOKININ-PANCREOZYMIN AND IONOPHORE A23187. (Eng.) Kanno, T.; Saito, A.; Sato, Y. (Faculty Veterinary Medicine, Hokkaido Univ., Sapporo, 060, Japan). *J. Physiol. (Lond.)* 270(1):9-28; 1977.

The roles of sodium and calcium ions in stimulus secretion coupling were analyzed in the isolated and perfused rat pancreas. Partial replacement of NaCl with LiCl produced a diminution in both amylase output and pancreatic juice flow induced by cholecystokinin-pancreozymin (CCK-PZ, 5 mU/ml), and almost normal responses were usually regained immediately after the reintroduction of a standard concentration of NaCl. Nearly total replacement of NaCl with LiCl caused an almost complete inhibition of the responses, although 25 mM $NaHCO_3$ and 1 mM NaH_2PO_4 were still present, and only partial recovery was obtained after the reintroduction of a standard concentration of NaCl. A quantitative relation was found between the amount of amylase released by CCK-PZ and the $[Na^+]_0$ over the range 26-157 mM in the presence of 2.5 mM Ca. A similar relationship was also observed when $[Ca^{2+}]_0$ was decreased to 1.0 mM, but the responses were reduced to about one-half of those observed with 2.5 mM Ca. The cellular mechanism of CCK-PZ-induced amylase output may involve the dominant activity of a complex composed of a carrier molecule bearing one Ca and four Na molecules, if there is no interaction be-

tween Na^+ and Li^+. A quantitative relation was also found between the amount of pancreatic juice flow stimulated by CCK-PZ and $[Na^+]_0$ over the range 26-157 mM, in the presence of 1.0 or 2.5 mM Ca. A similar quantitative relationship was found between the amount of amylase released by Ca ionophore A23187 and $[Na^+]_0$ in the presence of 2.5 or 5.0 mM Ca. This may also involve the dominant activity of a complex composed of a carrier molecule bearing one Ca and four Na molecules, if there is no interaction between Na^+ and Li^+.

0113 THE EFFECT OF SOMATOSTATIN ON EXOCRINE PANCREAS SECRETION IN THE DOG STIMULATED BY PANCREOZYMIN-SECRETIN, OR BY A TEST MEAL. (Ger.) Kayasseh, L.; Stalder, G. A.; Gyr, K.; Rittmann, W. W.; Girard, J. (Medizinische Universitatspoliklinik, Hebelstrasse 1, CH-4056 Basel, Switzerland). *Schweiz. Med. Wochenschr.* 107(15):541-542; 1977.

The effect of somatostatin (SST) on the exocrine pancreas of dogs with pancreatic and gastric fistulae was investigated during stimulation by pancreozymin-secretin or by a test meal. Chronic pancreatic and gastric fistulae were made following ligation of the minor pancreatic duct. The exocrine pancreas was infused with 2 IU of secretin-pancreozymin for 150-180 min, and SST was first given as a bolus injection (3.5 µg/kg) followed by infusion (3.5 µg/kg/hr). In another experiment, the pancreas was stimulated by means of an intragastrically administered Lundh test meal (5 ml/kg). SST caused a significant inhibition of stimulated enzyme secretion of the exocrine pancreas; protein secretion into the pancreatic juice was reduced by 54% (273.7 ± 57.2 to 125.8 ± 4 mg/hr) and amylase by 57.7% (42,115.9 ± 8,928.8 to 17,817 ± 5,098 U/hr). However, bicarbonate secretion and volume did not change significantly. The bicarbonate concentration was reduced by 8.1% (133.8 ± 1.7 U/hr to 123 ± 3.9 U/hr). The bilirubin content of the duodenal juice also decreased significantly (by 88%) during SST treatment (from 32 to 3.8 mg%). Similarly in the Lundh test meal trial, SST caused a 71% decrease in trypsin concentration (23 ± 3.3 to 6.7 ± μEqH^+/ml/min) and a 57.2% decrease in amylase level (208.5 ± 27.3 to 89.2 ± 18.4 U/ml). The total volume of duodenal juice was reduced by 51.2% (213 ± 29.6 to 104 ± 36.5 ml/hr).

0114 INFLUENCE OF SECRETIN ON CAERULEIN STIMULATED PANCREATIC SECRETION OF THE RAT. (Eng.) Linari, G.; Linari, M. B. (Inst. Medical Pharmacology I, Univ. Rome, 0185 Rome, Italy). *Pharmacol. Res. Commun.* 9(7):633-641; 1977.

The pancreatic dose-response relationship to graded doses of cerulein was studied in female Wistar rats under acute fistula conditions. Cerulein (3.75-101.25 ng/kg/min, i.v.) was infused over a continuous secretin background (6 U/kg/hr). In comparison with rats that did not receive secretin, the flow of juice was significantly ($p<0.01$) increased in response to all doses of the peptide, but only the rate of 101.25 ng/kg/min produced a significant increase of amylase output. A good correlation between flow and enzyme secretion was seen. Amylase concentrations were about 30% lower than in animals not infused with secretin. The secretin background determined an increase of the calculated maximal response of amylase secretion to cerulein and a decrease of the ED_{50} value of the peptide. These studies demonstrate that the kinetics of the pancreatic response to cerulein are modified by a secretin background.

0115 AMINO ACID TRANSPORT IN THE RAT EXOCRINE PANCREAS. II. INHIBITION BY LANTHANUM AND TETRACAINE. (Eng.) Bieger, W.; Peter, S.; Volkl, A.; Kern, H. F. (Dept. Anatomy, Univ. Heidelberg, Heidelberg, W. Germany). *Cell Tissue Res.* 180(1):45-62; 1977.

The effect of increasing concentrations of lanthanum and tetracaine on the accumulation of labeled neutral amino acids in the free intracellular pool of isolated rat pancreatic lobules was studied *in vitro*. The two calcium antagonistic agents caused severe disturbances in the secretory process of the exocrine pancreas, including inhibition of the rate of protein synthesis and exocytosis. The former effect resulted mainly from the inhibition of amino acid transport. Lanthanum (up to 1 mM) inhibited the transport of different species of amino acids in an unspecific way, whereas tetracaine interfered specifically with the Na^+-dependent transport system for neutral amino acids (^{14}C-α-aminoisobutyric acid). Na^+-independent transport of neutral amino acids (^3H-leucine) was not affected. Transport inhibition was correlated to the activity of the Na^+,K^+-ATPase system, which was measured in isolated plasma membrane fractions. At higher concentrations (5-10 mM), some uptake of lanthanum into the cells by limited endocytosis was observed. At lower concentrations, lanthanum seemed to bind exclusively to certain components of the plasma membrane, mainly at the lateral and basal cell surface. Even at 5-10 mM, no binding to the apical surface occurred. Similarly, lanthanum was not bound to the limiting membrane of isolated zymogen granules, but the mitochondria in the same fraction showed considerable binding affinity. The action of lanthanum and tetracaine on membrane carrier systems did not affect the interior organization of the plasma membrane. Particle density and distribution in freeze-fracture replicas as well as the submembrane microfilamentous-microtubular system and the junctional elements remained unaffected.

0116 EVIDENCES FOR DUODENOPANCREATIC REFLEXES AND ANTI-CCK FACTOR WITH LIDOCAINE INFUSED INTRAVENOUSLY AND SPRAYED TOPICALLY ON PANCREATIC PAPILLA IN NONALCOHOLIC AND ALCOHOL-FED DOGS. (Eng.) Tiscornia, O. M.; Sarles, H.; Voirol, M.; Levesque, D.; Dzieniszewski, J.; Palasciano, G.; *et al.* (Unite de Recherches de Pathologie Digestive, INSERM, 46 Chemin de La Gaye, 13009 Marseilles, France). *Am. J. Gastroenterol.* 66(3):221-240; 1976.

The effect of lidocaine, injected i.v. or applied topically to the duodenal pancreatic papilla, on secretin-induced pancreatic secretion was studied in 14 duodenal fistula dogs, 4 of which had been alcohol-fed for 2 yr. Lidocaine was also applied directly to the papilla or injected into dogs that had received secretin and cholecystokinin (CCK). Pancreatic secretion was measured by flow rate during 20 min, bicarbonate concentration and output, and protein concentration and output. Lidocaine applied topically to the duodenal pancreatic papilla inhibited pancreatic secretion but had the opposite effect in dogs administered CCK plus secretin. Chronic alcohol feeding enhanced the lidocaine-induced rise in pancreatic secretion in dogs given CCK and secretin. When lidocaine was injected i.v., no significant changes in pancreatic secretion were observed in nonalcoholic dogs. Effects similar to those of topically applied lidocaine were observed with i.v. lidocaine in alcoholic and nonalcoholic superimposed on CCK and secretin stimulation did not prevent and actually elevated the i.v. lidocaine-induced pancreatic secretion changes. It is postulated that lidocaine acts directly on the papilla by interrupting duodenopancreatic reflexes. Lidocaine may modify pancreatic secretion in the CCK- and secretin-stimulated animals by depressing an anti-CCK factor secreted by the small intestine.

0117 ROLE OF THE PANCREAS IN STABILITY OF MITOCHONDRIAL FUNCTION IN HEMORRHAGIC SHOCK. (Eng.) Sato, M.; Yamamoto, M.; Ozawa, K.; Honjo, I. (Dept. Surgery, Kyoto Univ., Kyoto, Japan). *J. Surg. Res.* 23(1):19-24; 1977.

The role of the pancreas in adenine nucleotide and mitochondrial metabolisms during hemorrhagic shock was studied in male Wistar rats. Pancreatectomized rats showed more rapid depletion of energy stores during hemorrhagic shock than did nonpancreatectomized animals. In pancreatectomized rats the oxidative and phosphorylative activities of the mitochondria decreased markedly to about 40% of the preshocked levels at 1 hr after hemorrhagic shock, although those in control rats remained almost unchanged. The decreases in mitochondrial oxidative and phosphorylative activities and hepatic energy charge levels in pancreatectomized rats were not restored by the reinfusion of the shed blood after 1-hr hemorrhagic shock. It is suggested that the pancreas plays an important role in hepatic energy metabolism during hemorrhagic shock, possibly due to maintaining the integrity of mitochondrial membrane.

0118 ULTRASTRUCTURAL AND FUNCTIONAL CHANGES IN PANCREATIC ACINAR CELLS DURING AUTOLYSIS. (Eng.) Nevalainen, T. J.; Anttinen, J. (Lab. Electron Microscopy, Univ. Turku, Kiinamyllynkatu 10, SF-20520 Turku 52, Finland). *Virchows Arch.* [*Cell Pathol.*] 24(3):197-207; 1977.

Ultrastructural changes during *in vitro* autolysis were studied in the rat pancreas. Tissue samples were incubated at 18-20 C in a moist atmosphere for 0, 0.5, 1, 3, 6, 12, and 24 hr. The disintegration of acinar cells began by swelling of various cell compartments and gradual breakdown of cell membranes. Zymogen granules remained morphologically intact for at least 3 hr. There were no signs of increased autophagic activity during the observation period. Myelin figures and other membranous remnants of disintegrated cells, together with individual cells and cell organelles whose morphology was relatively well preserved were seen even after 24 hr incubation. The secretory response of acinar cells to pancreozymin stimulation, as measured by amylase release into the incubation medium *in vitro*, decreased progressively, approaching zero, during the 12-hr autolysis. No active trypsin was detected in the tissue samples during the 24-hr observation time. It is concluded that, during hypoxic autolysis at room temperature between 18-20 C *in vitro*, acinar cell disintegration results from breakdown of cellular membranes and that autophagocytosis is not involved, that most zymogen granules remain morphologically intact, even at the time when cell membranes show evidence of damage, that no trypsin activation takes place in the tissue during hypoxic autolysis, and that the acinar cells are capable of responding to secretory stimulation for 3-6 hr after removal of the tissue from the experimental animal.

0119 PANCREATITIS FOLLOWING THE INTRADUCTAL INJECTION OF PARTIALLY PURIFIED ENTEROKINASE IN DOGS. (Eng.) Hammond, J. B.; Mann, N. S. (Veterans Admin. Lakeside Hosp., 333 E. Huron, Chicago, IL 60611). *Am. J. Dig. Dis.* 22(3):182-188; 1977.

To determine whether enterokinase (EK) alone can cause the experimental pancreatitis (PT) produced by proximal and distal closure of bile duct ligated dogs, saline suspensions of partially purified EK and other test materials were injected into the duct of Wirsung of dogs. After 24 hr, their pancreases were examined, and the increment in serum amylase was estimated. Following 0.5% EK administration, both PT and hyperamylasemia ensued; hyperamylasemia without PT occurred when EK was inactivated by heat, administered with trypsin inhibitor, or administered in more dilute solution. The injection of trypsin inhibitor or of hog gastric mucin also led to hyperamylasemia, but not to PT. It is concluded that the PT observed was due to EK activity and that EK could contribute to the production of PT in the closed duodenal loop model. The hyperamylasemia observed in the absence of PT is unexplained, but appears to be related to the colloidal properties of the materials injected.

0120 INSULIN SECRETION AND PANCREATIC EXOCRINE SECRETION IN THE AGED. (Jpn.) Shimizu, Y.; Hiramatsu, K.; Miyake, K.; *et al.* (Okayama Univ. Sch. Medicine, Okayama, Japan). *Jpn. J. Geriatr.* 13(1):3-15; 1976.

0121 REPRODUCIBILITY OF VISCOSITY ASSESSMENT IN PURE HUMAN PANCREATIC JUICE [Abstract].

(Eng.) Tympner, F.; Rosch, W.; Domschke, W.; Demling, L. (Dept. Medicine, Univ. Erlangen-Nurnberg, Nurnberg, W. Germany). *Endoscopy* 9(3):191; 1977.

0122 POTENCY OF DES-HISTIDYL[1]-SECRETIN FOR PANCREATIC SECRETION IN DOGS [Abstract]. (Eng.) Solomon, T. E.; Beyerman, H. C.; Grossman, M. I. (Veterans Admin. Wadsworth Hosp. Center, Los Angeles, CA). *Clin. Res.* 25(4):574A; 1977.

0123 MECHANISMS OF HUMAN PANCREATIC POLYPEPTIDE (HPP) RELEASE [Abstract]. (Eng.) Taylor, I. L.; Richardson, C. T.; Feldman, M.; Walsh, J. H. (Veterans Admin. Wadsworth Hosp., Los Angeles, CA). *Clin. Res.* 25(4):574A; 1977.

0124 EFFECT OF BOVINE PANCREATIC POLYPEPTIDE (BPP) INFUSIONS ON GASTRIC AND BILIARY FUNCTION IN MAN [Abstract]. (Eng.) Greenberg, G. R.; McCloy, R. F.; Chadwick, V. S.; Adrian, T. E.; Baron, J. H.; Bloom, S. R. (Hammersmith Hosp., Ducane Rd., London, England). *Gut* 18(11): A978; 1977.

0125 PP IN THE ASSESSMENT OF PANCREATIC EXOCRINE FUNCTION [Abstract]. (Eng.) Adrian, T. E.; Besterman, H. S.; Mallinson, C. N.; Bloom, S. R. (Hammersmith Hosp., Ducane Rd., London, England). *Gut* 18(11):A982; 1977.

0126 CHOLINERGIC NERVOUS CONTROL OF THE PANCREAS. (Fre.) Tiscornia, O. M. (INSERM, U-31, 46, chemin de la Gaye, F 13009 Marseille, France). *Biol. Gastroenterol. (Paris)* 9(Suppl. 3): 255-275; 1976.

0127 MORPHOLOGICAL BASIS OF EXPERIMENTAL PANCREATITIS. (Rus.) Kiseleva, A. F.; Keisevich, L. V.; Degtiareva, L. V.; Diachenko, V. V. (Kiev Medical Inst., Kiev, USSR). *Vrach. Delo* (9):100-104; 1976.

See also, 0009, 0106, 0226, 0243, 0274, 0280, 0506, 0507, 0515, 0519, 0524, 0533, 0652.

SECRETION AND METABOLISM

Hepatobiliary Tract

0128 EFFECT OF FASTING ON THE BIOCHEMICAL PROPERTIES OF PERFUSED RAT LIVER. (Eng.) Lee, D.; Clark, D. G. (Univ. Otago Medical Sch., P.O. Box 913, Dunedin, New Zealand). *J. Surg. Res.* 23(1):43-49; 1977.

Livers from male Wistar rats that had been either fed *ad libitum* or fasted for 48 hr were perfused with bovine RBC in Krebs-Ringer buffer containing 3% bovine serum albumin, and their function was studied. There was little difference in the ability of livers from fed or fasted rats to produce bile. Livers from fed rats maintained their initial rate of urea synthesis (1.5 μmol/min) for 60 min, but livers from fasted rats maintained this rate for only 20 min. At the end of these periods, urea synthesis fell to 0.8 μmol/min and 0.6 μmol/min for livers from fasted and fed rats, resp. Potassium was released into the acellular fraction of the perfusate at a rate of 0.3 μEq/min 60 min after the start of perfusion of livers from fed rats and 20 min after the start of perfusion for fasted rats. Compared with unperfused controls, perfusion for 3 hr had little effect on the latency (62.2 ± 3.1% versus 60.5 ± 2.3% for controls) and sedimentability (62.6 ± 2.9% versus 68.0 ± 1.2% for controls) of β-glucuronidase or on the sedimentability (69.1 ± 2.2% versus 69.7 ± 0.7% for controls) of acid phosphatase in liver homogenates from fed rats. There was a significant decrease in latency of acid phosphatase (66.6 ± 1.7% versus 75.8 ± 1.8% for controls) in liver homogenates from fed rats. Perfusion of livers from fasted rats resulted in marked and statistically significant falls in the latency (46.4 ± 3.0% versus 72.8 ± 1.2% for controls) and sedimentability (56.4 ± 2.5% versus 78.1 ± 1.8% for controls) of acid phosphatase and in the sedimentability (53.4 ± 2.3% versus 65.7 ± 1.5% for controls) of β-glucuronidase. The fall in latency of β-glucuronidase (55.5 ± 1.7% versus 57.0 ± 1.9% for controls) was not statistically significant. It is concluded that the livers from fasted rats are less able to tolerate the stress of perfusion *in vitro* than livers from rats fed *ad libitum*.

0129 A MORPHOMETRIC STUDY OF THE INHIBITION OF AUTOPHAGIC DEGRADATION DURING RESTORATIVE GROWTH OF LIVER CELLS IN RATS RE-FED AFTER STARVA-

TION. (Eng.) Pfeifer, U.; Bertling, J. (Pathologisches Institut der Universitat, Josef-Schneider-Str. 2, D-8700 Wurzburg, W. Germany). *Virchows Arch. [Cell Pathol.]* 24(2):109-120; 1977.

The number of autophagic vacuoles (AV) in the liver cells of rats re-fed after starvation was quantitated. In the liver parenchymal cells of adult male rats re-fed on the evening of the fifth day after a period of absolute starvation, a nearly complete absence of AV was found by the morphometric determination of the fractional cytoplasmic volume of AV. The mean value for that parameter increased only gradually during periods of re-feeding. The value was found to be in the range of the control values only on, or after, the fifth day of re-feeding. As in previous experiments, in the control animals the number of AV was again found to be dependent on a circadian rhythm with maxima during the light, and minima during the dark, periods. This rhythm reappeared in the period of re-feeding without a shift in phase. In the controls as well as in the re-fed animals the "segregated fraction" was highest for microbodies, intermediate for mitochondria and glycogen, but rather low for the remaining components of the cytoplasm. It is suggested that long-term inhibition of cellular autophagy plays an important role in the restorative cellular growth of the liver during the recovery from the atrophy induced by starvation.

0130 HEPATIC HEMODYNAMICS IN AN EXPERIMENTAL ANIMAL WITH PARTICULAR REFERENCE TO HEPATIC ARTERY FLOW. (Ger.) Thiel, H. (Med. Univ.-Klinik, Josef-Schneider-Strasse 2, 87 Wurzburg, W. Germany). *Fortschr. Med.* 95(27):1741-1744; 1977.

Hepatic and systemic hemodynamics were studied in healthy rats and rats with liver diseases, with particular emphasis on the regulation mechanism of the hepatic artery and compensatory changes of hepatic arterial blood flow. The trials were performed with the cardiac output fractionation technique in animals with portal hypertension (brought about in one of three ways: prehepatically after protracted occlusion of the portal vein, intrahepatically following cirrhosis induced by choline-deficient diet or carbon tetrachloride, or by granulomatous hepatitis induced by SiO_2 particles). The experiments were also carried out in animals with galactosamine hepatitis, rats with portacaval shunt, in regenerating rat liver after partial hepatectomy, and in rats with artificial systemic endotoxemia. Hepatic arterial blood flow in both the normal and abnormal liver was regulated by a venovasomotor reaction (after portacaval end-to-side shunt) and systemically by autoregulation. As portal liver blood flow decreased, hepatic arterial flow increased, especially after portacaval end-to-side shunt. However, this increase did not fully compensate for the diverted portal venous blood flow. A significant rise in circulating blood volume as a result of hemodilution was noted in protracted occlusion of the portal vein, in CCl_4-induced cirrhosis, and SiO_2-induced granulomatous hepatitis. A definite relation was noted between the elevation of circulating blood volume and the extent of portacaval shunt volume. Cardiac output and partial circulating times also showed a close relation to the extent of portacaval shunt flow.

0131 THE HEPATIC ARTERY: SUBSERVIENT TO HEPATIC METABOLISM OR GUARDIAN OF NORMAL HEPATIC CLEARANCE RATES OF HUMORAL SUBSTANCES. (Eng.) Lautt, W. W. (Dept. Physiology, Univ. Saskatchewan, Saskatoon, Saskatchewan, Canada). *Gen. Pharmacol.* 8(2): 73-78; 1977.

Studies indicating that local tissue demands (at least within physiological limits) do not affect hepatic arterial flow and that the hepatic artery is not affected by metabolites or local hypoxia are reviewed. With the use of an electromagnetic flowmeter, a method by which uptake or output of bloodborne substances can be determined simultaneously with hemodynamic measurements of the intact liver was developed. O_2 delivery to cat liver was reduced to 68% of control; in spite of a marked reduction of O_2 content and O_2 pressure in the venous blood, no arterial vasodilation was seen, although the arteries supplying the gut did dilate. Thus, the hepatic artery is not subservient to local hepatic needs, at least within the limits examined. Other studies have shown that decreases in hepatic metabolism are not accompanied by decreased hepatic blood flow. It is suggested that the hepatic artery serves to maintain blood flow *per se* constant (as opposed to O_2 delivery). The significance of constant hepatic blood flow is indicated by the fact that altered hepatic blood flow produces altered rates of hepatic clearance of many substances. By compensating for wide fluctuations in portal venous flow, the hepatic artery prevents equally wide fluctuations in hepatic clearance rates of endogenous substances. The constancy of hepatic clearance rates thereby prevents wide swings in plasma hormone levels that would otherwise be finely controlled only by altered rates of synthesis and release from the endocrine glands. The hepatic artery is thus proposed to be subservient not to the liver but rather to the needs of the entire organism, and it is an essential component in the maintenance of stable levels of substances cleared by the liver.

0132 ENZYMATIC ELIMINATION OF SUBSTRATES FLOWING THROUGH THE INTACT LIVER. (Eng.) Bass, L.; Keiding, S.; Winkler, K.; Tygstrup, N. (Dept. Mathematics, Univ. Queensland, Australia). *J. Theor. Biol.* 61(2):393-409; 1976.

Enzymatic elimination of a class of substrates by the liver is theoretically analyzed in terms of a convective model of the hepatic microcirculation carrying the substrates. The class of elimination reactions considered is represented by the irreversible phosphorylation of D-galactose by galactokinase; D-galactose is important as a diagnostic substance in liver disease. A model liver is considered that consists of a large number of tubes

arranged in parallel, each corresponding to one sinusoid with its unidirectional blood flow and its adjoining parenchymal cells containing enzyme. The total blood flow through the liver and the total maximum elimination rate (Vmax) receive equal contributions from all sinusoids. The half-saturation concentration of the relevant substrate (Km) is the same throughout the model. The convective model establishes connections between the inflow and outflow substrate concentrations, the total blood flow, and the overall substrate elimination rate. The two kinetic parameters Vmax and Km are involved as constants to be inferred for each liver from the above four observable parameters. The model is used to predict the time course of galactose concentration in the hepatic artery and vein of a patient with liver disease after a single i.v. injection of galactose. The model shows a capacity of the liver to compensate in part for the effects of disease (loss of Vmax) by an increase in biochemical efficiency. The model is also tested and validated on four prolonged near-steady states that were established on a perfused isolated pig liver by controlled galactose infusions.

0133 QUANTITATION OF PATHWAYS OF ETHANOL METABOLISM. (Eng.) Havre, P.; Abrams, M. A.; Corrall, R. J. M.; Yu, L. C.; Szczepanik, P. A.; Feldman, H. B.; *et al.* (Case Western Reserve Univ., Sch. Medicine, Cleveland, OH 44106). *Arch. Biochem. Biophys.* 182(1):14-23; 1977.

A method for estimating the sum of the contributions to ethanol oxidation by the microsomal ethanol-oxidizing system (MEOS) and by catalase in the intact liver cell is described. It depends upon a comparison of the fate of the *R* hydrogen of ethanol and the hydrogen bound to carbon-2 of sorbitol under identical conditions. One limitation of this method is that it is based on the assumption that isotopic discriminations are similar for alcohol dehydrogenase, MEOS, and catalase. For either or both the MEOS and catalase oxidations, there may be much greater isotopic discrimination than for alcohol dehydrogenase, so that ^3H conversion to ^3H_2O by either of these pathways finds relatively little expression in the data. Using incubation of liver slices from rat and monkey at an ethanol concentration of 3 mg/ml, and from rat at 1 mg/ml, alcohol dehydrogenase catalysis was found to account for an average of 89% or more of the initial metabolism of ethanol. The stereospecificity of the sorbitol dehydrogenase-catalyzed reaction was shown to be of the A type in the rat. Sorbitol oxidation in the intact liver cell was found to be irreversible.

0134 METABOLIC CONSEQUENCES OF ALCOHOL CONSUMPTION. (Eng.) von Wartburg, J. P. (Medizinisch-chemisches Institut der Universitat Bern, Buhlstrasse 28, CH-3012 Bern, Switzerland). *Nutr. Metab.* 21(1/2/3):153-162; 1977.

The metabolism of alcohol is reviewed. The metabolic effects of alcohol are closely related to organic as well as societal aspects of alcoholism. Individual differences in the biologic sensitivity to ethanol are thought to represent important features underlying these afflictions. The results of recent biomedical research provide evidence for a biochemical individuality with respect to the enzymes involved in alcohol metabolism. Human liver alcohol dehydrogenase, the principle ethanol oxidizing enzyme in man, exists in multiple molecular forms (isoenzymes). In addition, a genetic polymorphism (atypical variant) is observed. Large racial differences are found for the prevalence of the variant enzyme, which is characterized by a high specific activity. Possible relationships of the individual isoenzyme pattern of alcohol dehydrogenase and of other alcohol metabolizing enzymes to the nature and extent of metabolic effects of ethanol are discussed.

0135 METABOLIC AND HEPATIC EFFECTS OF ALCOHOL. (Eng.) Isselbacher, K. J. (Harvard Medical Sch., Boston, MA 02114). *N. Engl. J. Med.* 296 (11):612-616; 1977.

Effects of alcohol on carbohydrate, protein, amino acid, and lipid metabolism are discussed and some disorders associated with alcoholism are reviewed. Alcohol inhibits gluconeogenesis and can lead to hypoglycemia if there is a substantial depletion of liver glycogen. Galactose oxidation is also inhibited by alcohol, although this is not usually clinically apparent. Albumin and transferrin synthesis are inhibited by alcohol, while lipoprotein synthesis is stimulated; this latter effect may contribute to the hypertriglyceridemia associated with alcohol ingestion. The major effects of alcohol ingestion on lipid metabolism are hypertriglyceridemia and fatty liver, resulting from the stimulation of serum and liver triglycerides. The increased NADH-NAD ratio induced by alcohol ingestion leads to increased lactate production, resulting in hyperuricemia; increased delta-aminolevulinic acid synthesis, leading to increased porphobilinogen production; and decreased citric acid cycle activity. Other effects induced by alcohol include increased acetaldehyde levels in the blood and tissues, decreased serum magnesium and phosphate, increased catecholamine release leading to decreased beta-adrenergic-receptor sensitivity, increased oxygen consumption, and ketoacidosis. The pathogenesis of ketoacidosis may involve hormonal factors and enhanced generation of NADH. The ability of alcohol to decrease the intestinal absorption of sugars, amino acids, calcium, folic acid, vitamin B_{12}, and other nutrients may contribute to the impairment in nutrition that is frequently present in alcoholic patients. Recent observations may explain the morphological alterations seen in alcoholic hepatitis. Hepatocyte ballooning may involve hypoxemia (produced by the hypermetabolic state induced by alcohol), alcohol-induced retention of export proteins by the liver, and the antitubular action of alcohol. Collagen probably plays an important role in the development of cirrhosis.

0136 ETHANOL METABOLISM BY A TRANSPLANTABLE HEPATOCELLULAR CARCINOMA: ROLE OF MICROSOMES AND MITOCHONDRIA. (Eng.) Cederbaum, A. I.; Becker, F. F.; Rubin, E. (Mount Sinai Sch. Medicine, City Univ. New York, New York, NY 10029). *J. Biol. Chem.* 251(17):5366-5374; 1976.

Ethanol metabolism was studied in rat hepatocellular carcinoma 252 (HC-252), and the effects of the activity of alcohol dehydrogenase, of the activity of the substrate shuttles, of the ability of the mitochondria to oxidize reducing equivalents, and of the activity of the microsomal ethanol-oxidizing system on this process were investigated. Ethanol metabolism in slices or homogenates of the transplantable carcinoma was 50-60% of the rate found in host liver slices or homogenates when they were expressed per gram of tissue wet weight and 70-80% of the liver when the rates were expressed per milligram of tissue protein. At 10 mM ethanol, the activities of alcohol dehydrogenase in tumor and liver supernatants were comparable. Tumor microsomes did not oxidize ethanol in the presence of a NADPH-generating system, indicating the absence of the microsomal ethanol-oxidizing system and catalase-mediated peroxidation of ethanol. The HC-252 microsomes were contaminated with catalase, and acetaldehyde production occurred in the presence of a H_2O_2-generating system (xanthine oxidase). The virtual absence of ethanol oxidation and drug metabolism (aminopyrine demethylase and aniline hydroxylase) in HC-252 microsomes may be due to the low activities of NADPH-cytochrome c reductase, NADPH oxidase, and NADPH-dependent oxygen uptake. Microsomal oxidation of ethanol was present in the well-differentiated tumor, Morris hepatoma 5123C, while activity was negligible in microsomes from Morris hepatoma 7288CTC, a less differentiated tumor. Microsomal NADPH oxidase was present in the well differentiated tumor 5123C, but was lacking in the less differentiated tumor 7288CTC. Several microsomal, mitochondrial, and cytosolic properties of HC-252 were similar to those of Morris hepatoma 7288CTC, but differed from those of the more differentiated 5123C tumor and normal liver. The content of mitochondrial protein in HC-252 was only 25% that of liver, and oxygen consumption was only 28% that of the liver. When corrected for the mitochondrial protein content, oxygen uptake in tumor HC-252 and liver homogenates was comparable. Isolated tumor and liver mitochondria displayed comparable state 4 and 3 rates of oxygen consumption with succinate and glutamate as substrates. The activities of the reconstituted malate-aspartate and α-glycerophosphate shuttles were only slightly lower in isolated HC-252 mitochondria than in liver mitochondria. Antimycin inhibited alcohol metabolism, and pyruvate stimulated alcohol metabolism, ism, and pyruvate stimulated alcohol metabolism, much less in tumor slices than in liver slices, suggesting the presence of an augmented mitochondria-independent, cytosolic mechanism for oxidizing reducing equivalents in the tumor. These factors suggest that oxidation of NADH is the limiting factor in ethanol metabolism. Whereas, in the liver mitochondrial reoxidation is predominant, in HC-252, cytosolic reoxidation of NADH also plays a major role.

0137 THE INFLUENCE OF CHRONIC ETHANOL FEEDING TO RATS ON THE INTEGRITY OF LIVER MITOCHONDRIAL MEMBRANE AS ASSESSED WITH THE Mg^{2+}-STIMULATED ATPase ENZYME. (Eng.) Hosein, E. A.; Hofmann, I.; Linder, E. (Dept. Biochemistry, McGill Univ., Montreal, Quebec H3G 1Y6, Canada). *Arch. Biochem. Biophys.* 183(1):64-72; 1977.

Mg^{2+}-stimulated ATPase, used as an indicator of liver mitochondrial membrane damage by ethanol in Sprague-Dawley rats was compared in two mitochondrial preparations using substrate concentrations that gave near V activity. Results obtained with this enzyme using different temperature and substrate concentrations showed that ethanol treatment to rats had affected both the activity of this enzyme and the liver inner mitochondrial membrane. This was evidenced by the appearance of a phase-transition temperature at 32 C in Arrhenius plots of samples from the alcoholic liver but not from the control liver. It was observed that when the assay was carried out below 26 C, mitochondria from rats treated chronically with ethanol had diminished ATPase activity. In mitochondrial ghosts, chronic ethanol feeding caused a decrease in the phase-transition temperature observed in Arrhenius plots, usually associated with the lipid phase transition, from the normal value of 18.5 ± 0.2 to 15.8 ± 0.3 C in the alcoholic rats. A similar decrease in phase-transition temperature was also obtained with sonicated liver mitochondrial membrane preparations from chronically ethanol-fed rats compared with chow-fed controls. Throughout the entire range of temperatures used, the activation energies of the control and chronic alcoholic mitochondrial preparations were different. These results affirm that chronic ethanol treatment alters the liver mitochondrial membrane structure or enzyme microenvironment, which, in turn, may affect mitochondrial function.

0138 EFFECT OF TEMPERATURE ON THE ASSAY OF HEPATIC ALCOHOL DEHYDROGENASE. (Eng.) Carter, E. A.; Isselbacher, K. J. (Harvard Medical Sch., Boston, MA 02114). *Proc. Soc. Exp. Biol. Med.* 155(3):290-295; 1977.

The effect of assay temperature (T) on alcohol dehydrogenase (ADH) activity was investigated in rat and horse liver supernatants and purified horse liver, yeast, and human liver ADH. The assay mixture consisted of 50 mM glycine-NaOH buffer (pH 9.6), 50 mM ethanol, 1.5 mM NAD, and 0.1 ml of ADH-containing fraction. By raising the T from 25 to 60 C, the ADH activity of rat and horse liver supernatants was stimulated eight- and threefold, resp. At 55 C, purified human liver ADH activity was 2.5 times greater, and purified horse liver activity two times greater. Hepatic ADH was not stimulated if the heated samples were allowed to cool to room T before ethanol was added and the assay was then carried out at 25 C. Purified yeast ADH activity was inhibited at T's >45 C. The stimulating effect of increased T may involve changes in enzyme function. When 2-butanol and 2-butanone were used as substrates, ADH activity was enhanced equally in

both the forward and reverse directions. Increasing the T from 25 to 60 C increased the reduction of 17-β-hydroxy-5-β-androstan-3-one with NADH five-fold (from 13.9 to 63.3 nmol NADH oxidized/mg protein/min). Only at 50-60 C could ADH activity be detected in normal rat or human plasma. At 50 C, this activity (nmol NADH formed/ml plasma/10 min) increased from 30 to 85 and 118 at 1.5 and 3 hr, resp., after the administration of CCl_4 (2 ml/kg intragastrically) and from 8.9 to 14.4 after chronic ethanol feeding (5% of a liquid diet for 10 days). The increased sensitivity obtained by measuring plasma ADH activity at 50 C may make this assay useful for studying patients with liver injury.

0139 THE ROLE OF CYTOCHROME P-450 IN THE HYDROPEROXIDE-CATALYZED OXIDATION OF ALCOHOLS BY RAT-LIVER MICROSOMES. (Eng.) Rahimtula, A. D.; O'Brien, P. J. (Dept. Biochemistry, Memorial Univ. Newfoundland, St. John's, Newfoundland A1C 5S7, Canada). *Eur. J. Biochem.* 77(1):201-208; 1977.

The roles of the microsomes of cytochrome P-450 and of catalase in hydroperoxide-catalyzed ethanol oxidation was investigated in rat liver microsomes. When cumene hydroperoxide was the oxidizing agent, the pH optimum for microsomal ethanol oxidation was approximately 8.0, while the catalase-dependent reaction showed no pH optimum within the range of pH 6.5-9.0 but increased with increasing pH. Both catalase and the microsomes gave an apparent Km for ethanol in the region of 4-9 mM, but the microsomes gave an additional Km for ethanol in the region of 40-75 mM. In the microsomes, the two Km values were obtained even in the presence of 0.1 mM hydroxylamine, suggesting that the lower Km of 4-9 mM is not due to the small amount of catalase present in the microsomes. Catalase and microsomes also behaved differently in the presence of several inhibitors. In general, cytochrome P-450-specific agents (e.g., metyrapone and hexobarbital) inhibited the microsomal reaction, while they had no effect on the catalase-dependent reaction. Catalase-specific inhibitors (e.g., aminotriazole and hydroxylamine) inhibited the catalase-dependent oxidation but not the microsomal reaction. Phenobarbital pretreatment of the rats raised the specific activity of cumene hydroperoxide-dependent ethanol oxidation per milligram of microsomes by 7.2-fold. These results indicate that, in addition to catalase, another enzyme, presumably cytochrome P-450, is responsible for cumene hydroperoxide-dependent ethanol oxidation. When H_2O_2 replaced cumene hydroperoxide as the oxidizing agent, ethanol oxidation was not increased by phenobarbital induction and was inhibited by hydroxylamine but not by metyrapone. These results indicate that the microsomal system behaves more like catalase in H_2O_2-catalyzed oxidation, suggesting that the microsomal catalase is responsible for H_2O_2-dependent ethanol oxidation.

0140 INFLUENCE OF ETHANOL OXIDATION RATE ON THE LACTATE/PYRUVATE RATIO AND PHOSPHORYLATION STATE OF THE LIVER IN FED RATS. (Eng.) Poso, A. R.; Forsander, O. A. (Res. Lab. of the State Alcohol Monopoly, Alko, Box 350, SF 00101 Helsinki 10, Finland). *Acta Chem. Scand.* [*B*] 30(9):801-806; 1976.

To determine if the cytosolic redox changes induced by ethanol are reflected in the cytosolic phosphorylation state, the effect of the ethanol oxidation rate on the interaction between the phosphorylation state (the [ATP]/[ADP] x $[HPO_4]^{2-}$ ratio) and the redox state (the free $[NAD^+]$/[NADH] ratio) of the liver cytosol was studied in intact, fed, female Sprague-Dawley rats. The rate of ethanol oxidation was inhibited to different degrees; with 0, 0.1, 0.5, 1.0, and 2.0 mmol/g pyrazole, the ethanol oxidation rates were 4.6, 4.1, 3.6, 3.4, and 2.3 μmol/g liver (wet weight)/min, resp. The ethanol oxidation rate had no influence on the liver lactate level, but correlated significantly with the pyruvate level. Accordingly, a significant correlation was also found between the ethanol oxidation rate and the lactate/pyruvate ratio. The rate of ethanol oxidation correlated significantly with the liver 3-phosphoglycerate level. No change in the glyceraldehyde-3-phosphate level was found. No correlation was found between the ethanol oxidation rate and the glyceraldehyde-3-phosphate/3-phosphoglycerate redox couple. Ethanol administration slightly increased the liver ATP level, but the simultaneous administration of pyrazole eliminated this effect. Other adenine nucleotides and HPO_4^{2-} were not changed. The changes in the rate of ethanol oxidation had no effect on the phosphorylation state in the fed liver. It is assumed that in the fed liver the phosphorylation state is so well stabilized that the redox level has no influence.

0141 ATP METABOLISM IN AN ETHANOL INDUCED FATTY LIVER. (Eng.) Gordon, E. R. (Montreal General Hosp., Montreal, Canada). *Biochem. Pharmacol.* 26(13):1229-1234; 1977.

The mechanism responsible for the decrease in phosphate potential in the liver of rats fed ethanol for prolonged periods was determined. Changes in ATP utilization were assessed by measuring the activity of the $[Na^+ + K^+]$-activated ATPase system, and changes in ATP synthesis were assessed by determining the activity of the adenine nucleotide translocase (ANT) system. Since long-chain coenzyme A (CoA) derivatives of fatty acids regulate the translocation of ADP into mitochondria, their total cellular content and mitochondrial level were also determined. After 2 weeks on a liquid diet in which 36% of the caloric intake was ethanol, male Sprague-Dawley rats developed a fatty liver. This change was not associated with any significant alteration in hepatic ATP content or in the activity of the ANT system. After 4 weeks, excess neutral lipid in the liver still persisted, but it was associated with a marked increase in the level of long-chain CoA fatty acid derivatives. The hepatic ATP level was only 50% of normal, and the rate of translocation of ADP into the mitochondria was decreased. The activity of the ANT system was restored to normal

values if the long-chain CoA fatty acid derivatives were removed from the surface of the mitochondrial membrane by treatment of the preparation with defatted albumin. At this time, there was only a slight (15%) enhancement of the [Na^++K^+]-activated ATPase system. With the removal of ethanol from the diet, the ATP level quickly returned to normal. Changes in the hepatic ATP level correlated well with the activity of the ANT system, but they did not parallel changes in the activity of the Na^+ pump system. These findings indicate that the dominant feature leading to the decrease in the ATP/ADP x P_i ratio is a decreased synthesis of ATP.

0142 STUDIES IN THE ETHANOL-INDUCED CHANGES IN GLYCEROLIPID SYNTHESIS IN RATS AND THEIR PARTIAL REVERSAL BY n-(2-BENZOYLOXYETHYL)NORFENFLURAMINE (BENFLUOREX). (Eng.) Pritchard, P. H.; Brindley, D. N. (Univ. Hosp. and Medical Sch., Clifton Blvd., Nottingham NG7 2UH, England). *J. Pharm. Pharmacol.* 29(6):343-349; 1977.

The hypothesis that benfluorex (S-780) might reverse the ethanol-induced synthesis of hepatic triacylglycerol (TG) was tested, and the effects of ethanol and S-780 on lipid metabolism were studied. Male Wistar rats were treated daily for 5 days with S-780 (50 mg/kg), and 2 hr after the fifth treatment they were intubated with ethanol (5 g/kg) or with isocaloric glucose. S-780 did not alter the absorption or oxidation of [1-^{14}C]ethanol, nor did it decrease the concentration of ethanol and its water soluble metabolites in the tissues examined. The rate of hepatic glycerolipid synthesis was measured *in vivo* using [^{14}C]palmitate and [^{3}H]glycerol, and the accumulation of [^{14}C]palmitate in various tissues was determined 4 hr after i.v. injection. Ethanol treatment increased the relative rate of hepatic TG synthesis and decreased the synthesis of phosphatidylcholine (PC), phosphatidylethanolamine (PE), and phosphatidylserine plus phosphatidylinositol (PS + PI). The relative flux from phosphatidate (PA) to diacylglycerol (DG) and TG was increased. These changes were reflected in the accumulation of [^{14}C]palmitate in hepatic lipids and were obtained under conditions where the net rate of [^{14}C]palmitate oxidation to $^{14}CO_2$ was greater in those rats treated with ethanol instead of glucose. S-780 did not alter the oxidation rates over a 4-hr period. Ethanol treatment increased the accumulation of [^{14}C]palmitate in the PS + PI fraction of lung and decreased the proportion in PE. In the kidney, it increased the concentration of [^{14}C]palmitate in TG, DG, and PE and decreased the concentration in PC and PS + PI. Pretreatment of the rats with S-780 partially reversed the changes in palmitate accumulation in glycerolipids caused by ethanol. S-780 treatment also decreased the rate of hepatic TG synthesis from glycerol in the rats fed glucose. It increased the percentage recovery of [^{14}C]palmitate in the livers and kidneys of glucose-fed rats and decreased that in adipose tissue. This treatment decreased the proportion of [^{14}C]palmitate recovered in the TG of the kidneys and lungs of these rats and increased the proportion in PI + PS. The results show that S-780 interferes with the balance between phospholipid and TG synthesis *in vivo*. It produces a decrease in the synthesis and accumulation of TG in rats treated both with ethanol and glucose.

0143 LIPID PEROXIDATION IN ISOLATED HEPATOCYTES FROM RATS INGESTING ETHANOL CHRONICALLY. (Eng.) Remmer, H.; Albrecht, D.; Kappus, H. (Institut fur Toxikologie, Universitat Tubingen, Wilhelmstrasse 56, D-7400 Tubingen, W. Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 298(2):107-113; 1977.

The role of lipid peroxidation in ethanol-induced liver damage was investigated. Isolated hepatocytes from rats consuming ethanol (8.5 g/kg) daily produced twice as much malondialdehyde as liver cells from control animals. The release of LDH and the uptake of trypan blue in both control and test hepatocytes did not differ during the incubation period of 2 hr. Glutamate dehydrogenase (GLDH), however, was only set free into the medium from liver cells of ethanol-drinking rats, indicating that mitochondrial alterations are involved. Bromotrichloromethane ($CBrCl_3$) promoted lipid peroxidation in hepatocytes from ethanol-drinking rats to a much greater degree than in cells from control rats. The cell damage induced by $CBrCl_3$, indicated by a release of LDH and GLDH from the hepatocytes and by hepatic uptake of trypan blue was also much more pronounced in liver cells from ethanol-drinking animals. The stronger action of $CBrCl_3$ cannot be explained by an enhanced microsomal metabolism, because no increase of drug metabolizing enzymes was observed. The relatively low ethanol consumption did not influence body growth and liver weight and did not evoke any triglyceride accumulation. The normal balance between processes favoring lipid peroxidations and reactions protecting the liver cells seems to be shifted during alcohol intake to a state that promotes radical formation

0144 ROLE OF ACETALDEHYDE IN THE ETHANOL-INDUCED IMPAIRMENT OF GLYCOPROTEIN METABOLISM IN RAT LIVER SLICES. (Eng.) Sorrell, M. F.; Tuma, D. J.; Schafer, E. C.; Barak, A. J. (Veterans Admin. Hosp., 4101 Woolworth Ave., Omaha, NB 68105). *Gastroenterology* 73(1):137-144; 1977.

The mechanism responsible for ethanol-induced changes in the glycosylation of proteins was investigated in rat liver slices. Ethanol (5-50 mM) decreased [1-^{14}C]glucosamine and [^{14}C]leucine incorporation into total liver and microsomal proteins and greatly inhibited incorporation into medium (secretory) proteins. Pyrazole (4 mM) prevented the inhibition of glycoprotein synthesis by ethanol. Additions of methylene blue or pyruvate, which corrected the disordered redox state caused by ethanol oxidation, did not reverse the inhibitory effects of ethanol on glycosylation. Sorbitol also inhibited glycosylation; however, fructose similarly inhibited glycoprotein synthesis without altering the redox state. When low levels of acetaldehyde (0.12-0.70 mM) were maintained in the medium by means of infusion, a

significant inhibition of glycoprotein synthesis was observed. Acetate did not impair glycosylation. These data suggest that the ethanol-induced impairment of glycoprotein synthesis and secretion is a consequence of ethanol metabolism and further suggest that this inhibition is attributable to acetaldehyde formation rather than to the altered redox state resulting from ethanol oxidation.

0145 EFFECT OF A LOW-PROTEIN DIET ON ACETALDEHYDE METABOLISM IN RATS. (Eng.) Lindros, K. O.; Pekkanen, L.; Koivula, T. (Res. Lab. of the State Alcohol Monopoly, Box 350, SF-00101 Helsinki 10, Finland). *Acta Pharmacol. Toxicol.* 40(1):134-144; 1977.

The effect of dietary changes on liver alcohol and aldehyde dehydrogenase activities, as related to effects on ethanol and acetaldehyde metabolism, was investigated in male Sprague-Dawley rats. Feeding for 8 weeks on diets rich in carbohydrate or fat, but with normal protein content, induced minor changes relative to giving a balanced diet. A low-protein, high-carbohydrate diet (5% and 80% of calorie content, resp.) caused a significant ($p<0.001$) reduction of both alcohol and aldehyde dehydrogenase activities in the liver. The activity of the high K_m aldehyde dehydrogenase in the microsomal and soluble fractions appeared to be more reduced than that of the low K_m enzyme in the mitochondrial fraction. The blood acetaldehyde level was significantly ($p<0.001$) higher in rats on the protein-deficient diet in spite of their reduced ethanol elimination rates. The results suggest that protein deficiency deranges acetaldehyde metabolism and may thus increase the possible contribution of acetaldehyde to the effects caused by ethanol metabolism.

0146 KINETICS OF SHEEP-LIVER CYTOPLASMIC ALDEHYDE DEHYDROGENASE. (Eng.) MacGibbon, A. K. H.; Blackwell, L. F.; Buckley, P. D. (Dept. Chemistry, Massey Univ., Palmerston North, New Zealand). *Eur. J. Biochem.* 77(1):93-100; 1977.

The steady-state parameters of aldehyde oxidation were studied in sheep. Sheep liver cytoplasmic aldehyde dehydrogenase showed little pH dependence of V or k_{cat}. Some buffer anion effects were noted. The oxidation of aldehydes at pH 7.6 was quantitative, but irreversible. The initial velocity data indicated a sequential mechanism for the addition of substrates. Inhibition by NADH and the product analogue 2-bromo-2-phenylacetic acid, together with the known tight binding of NADH to the free enzyme, indicated an ordered mechanism with NAD^+ as leading substrate. Values for the rate of binding and dissociation of NAD^+ were obtained from the steady-state data. The values obtained were identical with those calculated from the data for the horse liver cytoplasmic enzyme. Close similarities are, in general, apparent for the horse and sheep liver cytoplasmic enzymes and with other tissue aldehyde dehydrogenases. Apparent substrate activation was observed with high concentrations of both acetaldehyde and propionaldehyde, a limiting value of 0.25/sec being obtained for k_{cat}. No isotope effect was observed on V using 1-^{2}H-propionaldehyde as substrate, suggesting that NADH release might be rate-limiting in the steady-state.

0147 ENZYMATIC DETERMINATION OF BILE ACIDS. THE NADP-SPECIFIC 7α-HYDROXYSTEROID DEHYDROGENASE FROM P. testosteroni (ATCC 11996). (Eng.) Skalhegg, B. A.; Fausa, O. (Dept. Biochemistry, Nyegaard and Co. A/S, Nycovn. 2, Oslo 4, Norway). *Scand. J. Gastroenterol.* 12(4):433-439; 1977.

Using the specific substrates 7α-hydroxy-5β-cholanic acid and 3α,7α,12α-trihydroxy-3,12-diacetyl-5β-cholanic acid methyl ester, crude extracts of *Pseudomonas testosteroni* grown on a steroid-containing medium were shown to exhibit 7α-hydroxysteroid:NADP-oxidoreductase activity. The enzyme was highly specific for NADP. Both free and conjugated 7α-hydroxy bile acids could act as substrates, but those of low polarity (few hydroxyl groups) seemed to be preferred, judging from initial reaction velocity studies. The optimal conditions appeared to be at pH 8.5-9.5 and at 25 C. Free sulfhydryl groups were essential for maximum catalytic activity, since the enzyme was inhibited by sulfhydryl-reacting substances such as *p*-chloromercuribenzoate and monoiodoacetic acid. The ketone-trapping agents, hydrazine hydrate and semicarbazide, acted as inhibitors. Upon dilution, the storage stability was severely reduced, but this effect was counteracted by the addition of glycerol at at concentration of 20% or more. Gel filtration experiments on Sephadex G-100 indicated that the molecular weight was about 80,000. Thus, when this enzyme is used for bile acid determination in different biological fluids, a 60-min incubation time at pH 8.5-9.5 and 25 C should be used. Sulfhydryl-reacting substances should be avoided, and EDTA should be included in the assay mixture to chelate heavy metal ions.

0148 EFFECTS OF CHRONIC CHOLERETIC INFUSIONS OF BILE ACIDS ON THE MEMBRANE OF THE BILE CANALICULUS: A BIOCHEMICAL AND MORPHOLOGIC STUDY. (Eng.) Nemchausky, B. A.; Layden, T. J.; Boyer, J. L. (SDTU Veterans Admin. Hosp. Hines, Hines, IL 60141). *Lab. Invest.* 36(3):259-267; 1977.

To determine whether choleretic infusions of bile acids modify the function or structure of the bile canalicular membrane, sodium taurocholate (NaTc) or dehydrocholate (DHC) was infused into male rats at a rate of 80 μmol/hr over an 18-hr period. Bile was collected by fistula, and phospholipid and cholesterol content was measured in bile, liver homogenates, and isolated liver plasma membranes (LPM) enriched in bile canaliculi. Na^-,K^+-ATPase, Mg^{2+}-ATPase, 5'-nucleotidase, and alkaline phosphatase activities were also measured in LPM. NaTc infusions enhanced cholesterol and phospholipid output in the bile (0.35 ± 0.12 and 3.40 ± 1.13 nmol/min/g before infusion for cholesterol and phospholipid, resp.; 1.17 ± 0.39 and 10.04 ± 3.94 nmol/min/g after in-

fusion; $p<0.001$ in both cases) in association with a significant increase in phospholipid in both LPM and liver homogenates. Although DHC infusions resulted in a comparable excretion of bile acid, phospholipid, and cholesterol, output in bile did not increase from control values, and the concentration of these lipids in LPM and liver homogenates also did not change. However, LPM Na^+, K^+-ATPase significantly increased after DHC infusions compared to NaTc-infused animals or controls. Neither bile acid altered the activities of Mg^{2+}-ATPase, 5'-nucleotidase, or alkaline phosphatase. Both bile acids increased the diameter of the bile canalicular lumen as assessed by scanning electron microscopy, and produced irregularities and outpouchings in the canalicular membrane. Diverticuli and loss of microvilli were most prominent with DHC infusions, but canalicular side branching and the density of microvilli either remained unchanged or increased following NaTc infusions. Although the morphologic findings are qualitative, the results indicate that chronic choleretic infusions of NaTc and DHC have divergent effects, not only on enzyme activities in LPM, but also on phospholipid composition and three-dimensional structure. These findings suggest that bile acids may alter biliary secretion through their osmotic effects and by modifying lipids and enzymes in the bile canalicular membrane.

0149 PROLONGED INDUCTION OF GERMFREE BILE ACID PATTERN IN CONVENTIONAL RATS BY ANTIBIOTICS. (Eng.) Gustafsson, B. E.; Gustafsson, J. A.; Carlstedt-Duke, B. (Dept. Germfree Res., Karolinska Institutet, Stockholm, Sweden). *Acta Med. Scand.* 201(3):155-160; 1977.

The duration of the germfree pattern of fecal bile acids was studied in male conventional rats following the administration of antimicrobial drugs. The rats received benzylpenicillin, neomycin, kanamycin, erythromycin, bacitracin plus neomycin, succinylsulfathiazole, or metronidazole (360,000 IU/kg, 180 mg/kg, 35 mg/kg, 360 mg/kg, 27,000 IU and 550 mg/kg, 360 mg/kg, or 20 or 100 mg/kg, resp.) by stomach tube for 5 days. Total fecal bile acids were analyzed in samples collected during periods of 3 days during the pretreatment period and during the 8 weeks following drug treatment. Metronidazole or succinylsulfathiazole had no or minor effects on the conventional bile acid pattern and the "bile acid index" (β-muricholic acid/deoxycholic acid) remained low. Benzylpenicillin, neomycin, or kanamycin induced a germfree bile acid pattern, i.e., increased the relative amounts of α- and β-muricholic acid in feces and eliminated deoxycholic acid and hyodeoxycholic acid from feces. The high bile acid index was normalized within 3 weeks after the termination of drug treatment, but the excretion of α- and β-muricholic acid was not normalized until a normal flora had been established by giving an enema with intestinal contents from intact, conventional rats. Treatment with erythromycin or bacitracin plus neomycin also produced a germfree bile acid pattern. In these cases, the bile acid index did not normalize after 5-8 weeks, and the excretion of the muricholic acids was not normalized until an enema with intestinal bacteria had been given. It is suggested that these long-lasting effects of antibiotics on the metabolism of bile acids in the intestinal tract should be considered after short-term antibiotic therapy in humans.

0150 DISSOLUTION RATES OF MODEL GALLSTONES IN HUMAN AND ANIMAL BILES AND IMPORTANCE OF INTERFACIAL RESISTANCE. (Eng.) Molokhia, A. M.; Hofmann, A. F.; Higuchi, W. I.; Tuchinda, M.; Feld, K.; Prakongpan, S.; *et al.* (Coll. Pharmacy, Univ. Michigan, Ann Arbor, MI 48109). *J. Pharm. Sci.* 66 (8):1101-1105; 1977.

Cholesterol monohydrate dissolution kinetics in human gallbladder bile were studied to determine the magnitudes of the *in vitro* dissolution rates, the rate resistances in human gallbladder bile, and the extent that the interfacial resistance is the rate determining factor. Dissolution rate studies also were conducted using human duodenal bile and animal bile (baboon, dog, and chicken gallbladder bile) for comparison. The dissolution rate resistance, R, ranged from 10^4sec/cm for chicken bile to 10^4-10^6 sec/cm for human bile. Interfacial resistance was the rate-determining factor for all results. Where chemical composition data were obtained, the R values for the human bile samples were consistent with predictions made from the simulated bile studies. In two human gallbladder specimens with low bile acid-lecithin molar ratios (i.e., 2.9 and 2.3), very high R values of 1.9×10^5 and 4.1×10^5 sec/cm were found. These values were in good agreement with the findings in the simulated bile studies and suggest that stone dissolution in patients with low bile acid-lecithin ratios may proceed very slowly, even when the bile is highly undersaturated with respect to cholesterol.

0151 EVALUATION OF A NEW MONKEY MODEL FOR THE REPEATED STUDY OF BILE SECRETORY PHYSIOLOGY. (Eng.) Wood, R. A. B.; Baker, A. L.; Hall, A. W.; Boyer, J. L.; Moossa, A. R. (Dept. Surgery, Univ. Chicago, 950 E. 59th St., Chicago, IL 60637). *Ann. Surg.* 185(3):349-355; 1977.

An unchaired monkey model for the repeated long-term study of biliary secretory physiology is reported. Postoperative management with cholangiography and long-term follow-up of weight gain and liver function were recorded. Evaluation was performed by repeated measurement, over a period of months, of bile flow and ^{14}C-erythritol clearance at three rates of i.v. taurocholate infusion in a random order. The response to secretin was tested at the final infusion rate of taurocholate for each experiment. The procedure was performed on nine monkeys, two died before the study as a result of surgical complications and two others were studied twice before they died. The remaining monkeys were studied 8, 7, 5, 3, and 2 times, resp., over a 4- to 7-month period. They demonstrated an

increase in bile flow and ^{14}C-erythritol clearance with increasing bile salt output. A dramatic choleresis over a wide range of bile salt excretion was noted with secretin, and a linear relationship between bile bicarbonate output and bile salt secretion rate was observed. The new model has several major advantages and gives higher bile flows than previously recorded in the chaired model.

0152 THE CONTRIBUTION OF THE EXTRAHEPATIC BILE DUCTS TO BILE FORMATION. (Eng.) Strasberg, S. M.; Petrunka, C. N.; Ilson, R. G. (Medical Sciences Bldg., Univ. Toronto, Toronto, Ontario M5S 1A8, Canada). *Can. J. Physiol. Pharmacol.* 54(5):757-763; 1976.

The contribution of the extrahepatic bile ducts to bile flow was examined in the rhesus monkey. Bile flow from the two sides of the liver was divided. The major extrahepatic bile ducts remained connected to only one side of the liver. Bile flow, and the concentration of ^{14}C-erythritol, bicarbonate, bile acid, and bilirubin in bile samples from the two sides of the liver were measured and compared in the fed state. An estimate of the net flow from the extrahepatic ducts was obtained from the ^{14}C-erythritol concentrations on the two sides of the liver and the bile flow rate on the side with the extrahepatic ducts. The ^{14}C-erythritol bile-plasma ratio was significantly lower in bile collected from the side with the extrahepatic bile duct (0.98 ± 0.03) than in bile from the other side of the liver (1.22 ± 0.07, $p<0.05$). About 10% of total hepatic bile flow originated in the extrahepatic bile ducts in the fed state. The bicarbonate ^{14}C-erythritol concentration ratio was significantly higher in bile from the side with the extrahepatic bile ducts ($p<0.05$). Bicarbonate-bile acid, and bicarbonate-bilirubin concentration ratios were also significantly higher in bile from the side of the liver with the extrahepatic ducts. The extrahepatic bile ducts have a physiologically significant role in the secretion of bile water. Bicarbonate is secreted in association with water in the extrahepatic ducts.

0153 THE SYNTHESIS AND EXCRETION OF NON-ERYTHROID BILIRUBIN IN THE DOG. (Eng.) Bergan, A.; Ostrem, T.; Enge, I. (Rikshospitalet, Oslo, Norway). *Scand. J. Gastroenterol.* 12(5): 605-609; 1977.

To investigate the synthesis and excretion of non-erythroid bilirubin, sequential samples of arterial, portal, and liver blood, as well as of bile and thoracic duct lymph, were analyzed for bilirubin and nonbilirubin radioactivity following the injection of labeled δ-aminolevulinic acid (^{14}C-ALA, 90-125 μCi, i.v.). After 2 min, only about 15% of the injected radioactivity was found in the plasma. In systemic blood, the first nonerythroid, radioactive bilirubin was detected within 10 min after the injection, and maximum radioactivity was found between 30 and 75 min after precursor injection. In those dogs in which simultaneous liver vein and aortic blood samples were taken, radioactive bilirubin was detected at the same time in the two vascular compartments. The concentration of radioactive bilirubin in the liver vein blood was, however, always lower than that in aortic blood. Lymphatic and bile bilirubin radioactivity was demonstrated at 30 min after the ^{14}C-ALA injection. The fact that the nonerythroid bilirubin is detected in plasma before it appears in the bile excludes enterohepatic circulation. Radioactive bilirubin in the lymph was not only later in appearance than that in the plasma, but also lower in concentration than that in the plasma of dogs with intact biliary passages; these results exclude the possibility that the liver lymphatics are of any importance in the transport of nonerythroid bilirubin from the liver to the blood. Evidence for a direct canalicular excretion of the nonerythroid bilirubin synthesized in the liver was found by comparing the curves for bilirubin and nonbilirubin radioactivity in the lymph and in the bile. In the bile, there was no time lag between nonbilirubin and bilirubin radioactivity. This excludes the possibility that the first bilirubin to appear in the bile is secondary to that in the blood, as was the case for lymph. Like the synthesis of bilirubin in the liver, the process of conjugation is known to be very rapid. These steps should not represent any substantial delay in bilirubin excretion. Thus, nonerythroid bilirubin synthesized in the liver seems to be excreted directly to the bile and does not recirculate through the blood.

0154 ENZYMATIC CONVERSION OF BILIRUBIN MONOGLUCURONIDE TO DIGLUCURONIDE BY RAT LIVER PLASMA MEMBRANES. (Eng.) Jansen, P. L. M.; Chowdhury, J. R.; Fischberg, E. B.; Arias, I. M. (Albert Einstein Coll. Medicine, Bronx, NY 10461). *J. Biol. Chem.* 252(8):2710-2716; 1977.

The conversion of bilirubin monoglucuronide to bilirubin diglucuronide was studied in subcellular fractions of rat liver. The highest specific activity for bilirubin diglucuronide formation occurred in a fraction highly enriched in plasma membranes. Studies of reaction stoichiometry and utilization of UDP-D-[^{14}C]glucuronic acid revealed that conversion of bilirubin monoglucuronide to bilirubin diglucuronide is not catalyzed by UDP-glucuronyltransferase, and results from transglucuronidation of bilirubin monoglucuronide, with formation of bilirubin diglucuronide and unconjugated bilirubin. When unconjugated bilirubin was infused i.v. into rats at rates exceeding the maximal hepatic excretory capacity, bilirubin monoglucuronide accumulated in serum, and bilirubin diglucuronide was found exclusively in bile as the predominant bilirubin metabolite. These results suggest that the formation of bilirubin diglucuronide occurs at the surface membrane of the liver cell. Conversion of bilirubin monoglucuronide to bilirubin diglucuronide may play a role in the transport of bilirubin glucuronides from liver to bile.

0155 ALTERATIONS PRODUCED IN THE ENDOPLASMIC RETICULUM BY CARBON TETRACHLORIDE. (Eng.) Smuckler, E. A. (Dept. Pathology, Univ. Washington, Seattle, WA 98195). *Panminerva Med.* 18(9/10):292-309; 1976.

Factors concerning carbon tetrachloride hepatotoxicity are reviewed. The evidence suggests that carbon tetrachloride is metabolized within the hepatocyte. This biochemical modification appears to take place within the endoplasmic reticulum, with a production of proximate toxins that modify cell function and, more specifically, appear to modify the function of the endoplasmic reticulum itself. Both modification of the anabolic function of the membrane and decreased capacity to form proteins, both for cell use and for export, is easily detectable. The injury is associated with a disaggregation of the ribosome-messenger amino acyl-transfer RNA complex, so that essentially "nascent" ribosomes are present. This is not associated with alteration of protein synthesis or with formation of messenger RNA or its transport or lifetime in the cytoplasm. The alteration within the cell sap and within the membrane surface suggests that the active product associated with the change is not localized solely to the endoplasmic reticulum. The modification of the activity of the membranous structure of the endoplasmic reticulum is also not uniform, but rather punctate. This is consistent with the notion that there are "hot spots" that seem to be affected by this toxin and are represented by modifications of these structures and functions. The question of whether the most severe and the most lasting changes are those that appear in the fatally injured cells has not been answered. Although this seems to be true for protein synthesis detected by autoradiography, no simple technique has been provided to carry out a similar study for the membranous components of the endoplasmic reticulum. While it is evident that these modifications are important and potentially meaningful for the maintenance of cellular function, an explanation of their interaction with cellular physiology has not been put forth. Furthermore, it is not known whether this model will provide detailed evidence for mechanisms potentially available and important in the modification of cell structures by other halogenated hydrocarbons.

0156 EARLY ALTERATIONS INDUCED BY CARBON TETRACHLORIDE IN THE LIPIDS OF THE MEMBRANES OF THE ENDOPLASMIC RETICULUM OF THE LIVER CELL: II. DISTRIBUTION OF THE ALTERATIONS IN THE VARIOUS LIPID FRACTIONS. (Eng.) Benedetti, A.; Casini, A. F.; Ferrali, M.; Comporti, M. (Istituto di Patologia Generale dell'Universita di Siena, Via Laterino 8, 53100 Siena, Italy). *Chem. Biol. Interact.* 17(2):167-183; 1977.

The distribution of the molecular alterations produced by CCl_4 in the various phospholipids of the microsomal membrane was studied to investigate the functional membrane damage caused by the halogenated hydrocarbon. Analysis of various phospholipid moieties showed that phosphatidyl ethanolamine (PE) has the highest diene conjugation absorption, followed by phosphatidyl serine (PS) and phosphatidyl inositol (PI); all show the "D" spot in chromatography. The phosphatidyl choline (PC) fraction contained only a minor absorption of conjugated dienes and no "D" spot. A decrease in arachidonic acid (20:4) content was found in PE ($p<0.05$), while no change in fatty acid pattern occurred in PC or neutral lipids. Quantitative estimation of fatty acid methyl esters of various lipid fractions was done by irreversible binding of ^{14}C from $^{14}CCl_4$ to various lipid classes. PS had the highest specific activity, while PC had the least irreversible binding of CCl_4 free radicals. Thin-layer chromatography of the PS fraction showed that only 11% of the radioactivity was associated with pure PS moiety, while the lipid fraction containing the remaining radioactivity could be an oxidation product. It is suggested that following CCl_4 intoxication, the free radical attack occurring in the PE fraction initiates an intense lipoperoxidation process. In conclusion, the various phospholipid fractions of liver microsomal lipids are affected differently by CCl_4 poisoning.

0157 CARBON TETRACHLORIDE ACTIVATION, LIPID PEROXIDATION, AND THE MIXED FUNCTION OXYGENASE ACTIVITY OF VARIOUS RAT TISSUES. (Eng.) Villarruel, M. C.; de Toranzo, E. G. D.; Castro, J. A. (Laboratorio de Quimica Bio-Toxicologica, CITEFA, Zufriategui y Varela, Villa Martelli, Pcia. de Buenos Aires, Argentina). *Toxicol. Appl. Pharmacol.* 41(2):337-344; 1977.

Carbon tetrachloride activation, lipid peroxidation, and mixed function oxidase activity were examined in the microsomal fraction of the liver and other tissues from male and female Sprague-Dawley rats. When fasted (12-14 hr) rats were inoculated i.p. with a solution of $^{14}CCl_4$ (27.5 mCi/mmol) in olive oil [14 x 10^6 disintegrations/min (dpm)/ml] in a volume of 5 ml/kg and were then sacrificed 3 hr later, the levels of irreversibly bound ^{14}C to microsomal lipids in male liver, female liver, and pregnant female liver were 1,225 ± 266, 729 ± 78, and 666 ± 330 x 10^3 dpm/g lipid, resp. The corresponding $^{14}CCl_4$ tissue concentrations were 5 ± 1, 5 ± 1, and 8 ± 4 dpm/g tissue, resp. The ratio between the irreversible binding of ^{14}C to microsomal lipids and the free $^{14}CCl_4$ concentration was significantly higher in these liver microsomal fractions than in other tissue microsomal fractions tested, including the kidney. Lipid peroxidation values in microsomal fractions from various tissues 3 hr after fasted rats were given 1 ml/kg of CCl_4 i.p. (expressed as absorbance at 243 nm x 1,000 for a solution containing 1 mg of microsomal lipid/ml) were 285 ± 30, 225 ± 20, and 251 ± 32 for the male liver, female liver, and pregnant female liver, resp., compared to control values of 176 ± 24, 132 ± 10, and 185 ± 42, resp. These differences were statistically significant ($p<0.01$). Lipid peroxidation did not occur after CCl_4 administration in the other tissues tested. There was generally a good correlation between CCl_4 activation in the liver (and other tissues) and cytochrome P-450

content, which measured 0.65 ± 0.23, 0.39 ± 0.06, and 0.09 ± 0.04 nmol/mg protein in the male liver, female liver, and pregnant female liver, resp. No correlation between CCl_4 activation and cytochrome c reductase activity was found. These results suggest that cytochrome P-450 is involved in CCl_4 activation and that irreversible binding of CCl_4 metabolites to cellular components, rather than lipid peroxidation, is responsible for the biochemical and/or ultrastructural tissue lesions due to CCl_4 intoxication.

0158 CIRRHOSIS OF THE LIVER IN RABBITS INDUCED BY A HIGH CHOLESTEROL DIET--AN EXPERIMENTAL MODEL. (Eng.) Gupta, P. P.; Tandon, H. D.; Ramalingaswami, V. (Dept. Veterinary Pathology, Punjab Agricultural Univ., Ludhiana, India). *Indian J. Med. Res.* 64(10):1516-1526; 1976.

Hepatic changes induced in rabbits fed different regimens of high cholesterol diet for a prolonged period of time were studied, and the possible use of these regimens for the study of fatty liver and its evolution into cirrhosis is described. The sequence of changes on continuous cholesterol feeding was: fatty change, which was initially centrilobular, but later became diffuse, with simultaneous accumulation of cholesterol in the parenchymal cells; diffuse portal fibrosis; and eventually "portal" cirrhosis of the monolobular type. Liver cell necrosis seemed to have contributed little to the pathogenesis of cirrhosis. The fibrosis was mainly related to the mesenchymal cell activity in the portal canals. There was no significant regression of changes when cholesterol was withdrawn from the diet after 8 weeks of feeding. One of four animals developed cirrhosis 20 weeks after withdrawal. The precise mechanism of fibrosis and cirrhosis is not understood, but excessive accumulation of cholesterol in the liver probably acts as a cirrhogenic factor. The morphological sequences of the cholesterol-induced portal cirrhosis provide a useful experimental model for the study of the mechanism of cirrhosis, using an endogenous agent, without mediation of nutritional deficiencies.

0159 LIVER DAMAGE FROM COCAINE IN MICE. (Eng.) Shuster, L.; Quimby, F.; Bates, A.; Thompson, M. L. (Tufts Univ. Sch. Medicine, Boston, MA 02111). *Life Sci.* 20(6):1035-1042; 1977.

The effects of cocaine on liver histology, serum transaminase activity, and *in vivo* drug metabolism were studied in mice. Four daily injections of cocaine hydrochloride (20 mg/kg) in $B6AF_1/J$ mice produced focal necrosis of liver parenchymal cells in the midzonal region and a fivefold increase in SGOT. Massive liver damage and marked SGOT elevation (from a normal level of 78 ± 8.5 Karmen U [KU]/ml to as high as 25,000 KU/ml) was observed after a single injection of 50 mg/kg cocaine-HCl. This damage led to increased sleep time after pentobarbital, and a decreased rate of pentobarbital metabolism *in vivo*. The results indicate that cocaine is a potent hepatotoxic agent in mice.

0160 UPTAKE AND DEGRADATION OF ^{125}I-LABELLED ASIALO-FETUIN BY ISOLATED RAT HEPATOCYTES. (Eng.) Tolleshaug, H.; Berg, T.; Nilsson, M.; Norum, K. R. (Sch. Medicine, Univ. Oslo, Blindern, Oslo 3, Norway). *Biochim. Biophys. Acta* 499(1): 73-84; 1977.

The uptake and degradation of ^{125}I-labeled asialofetuin was studied in isolated rat liver cells, and the distribution of this asialo-fetuin between hepatocytes and nonparenchymal liver cells was examined after i.v. injection. ^{125}I-labeled asialofetuin was taken up by a saturable process. Half-maximum uptake was seen at about 3×10^{-8} M asialofetuin. Nonparenchymal liver cells did not take up asialo-fetuin *in vitro*. The rate of uptake of asialo-fetuin exceeded the rate of degradation at all concentrations of asialo-fetuin tested. Asialofetuin consequently accumulated in the cells until the extracellular supply was exhausted. Asialofetuin degradation could be studied without concurrent uptake by incubating cells, previously exposed to asialo-fetuin, in asialo-fetuin-free medium. Degradation, as evidenced by increase in acid-soluble radioactivity, was inhibited by NH_4Cl and chloroquine. The change with time in the intracellular distribution pattern of radioactivity in cells that had been exposed to ^{125}I-labeled asialofetuin for 10 min was examined by means of differential centrifugation. Initially, the radioactivity was found mostly in the microsomal fraction. One hour after the exposure to labeled protein, the distribution pattern of radioactivity resembled that of the lysosomal enzyme β-acetylglucosaminidase. It is possible that asialo-fetuin digestion takes place in lysosomes.

0161 EFFECT OF DICHLOROACETATE AND GLUCAGON ON THE INCORPORATION OF LABELED SUBSTRATES INTO GLUCOSE AND ON PYRUVATE DEHYDROGENASE IN HEPATOCYTES FROM FED AND STARVED RATS. (Eng.) Claus, T. H.; Pilkis, S. J. (Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232). *Arch. Biochem. Biophys.* 182 (1):52-63; 1977.

The effects of dichloroacetate on the conversion of various labeled substrates into glucose, as well as its effect on pyruvate dehydrogenase activity, were studied in the isolated hepatocytes of fed and fasted rats. Dichloroacetate (2 mM) stimulated the conversion of [1-^{14}C]lactate to glucose in hepatocytes from fed rats. In hepatocytes from rats starved for 24 hr, where the mitochondrial NADH/NAD^+ ratio is elevated, dichloroacetate inhibited the conversion of [1-^{14}C]lactate to glucose. Dichloroacetate stimulated $^{14}CO_2$ production from [1-^{14}C]lactate in both cases. It also completely activated pyruvate dehydrogenase and increased flux through the enzyme. The addition of β-hydroxybutyrate, which elevates the intramitochondrial NADH/NAD^+ ratio, changed the metabolism of [1-^{14}C]lactate in hepatocytes from fed rats to a pattern similar to that seen in hepatocytes from starved rats. Thus, the effect of dichloroacetate on labeled glucose synthesis from lactate appears to depend on the mitochondrial oxidation-reduction state of the he-

patocytes. Glucagon (10 nM) stimulated labeled glucose synthesis from lactate or alanine in hepatocytes from both fed and starved rats and in the absence or presence of dichloroacetate, but the hormone had no effect on pyruvate dehydrogenase activity whether or not the enzyme had been activated by dichloroacetate. It appears, then, that pyruvate dehydrogenase is not involved in the hormonal regulation of gluconeogenesis. Glucagon inhibited the incorporation of 10 mM [1-^{14}C]pyruvate into glucose in hepatocytes from starved rats. Although this inhibition has been previously attributed to an inhibition of pyruvate dehydrogenase by the hormone, dichloroacetate did not prevent the inhibition of glucose synthesis, nor did glucagon alter the activity of pyruvate dehydrogenase in homogenates of cells that had been incubated with 10 mM pyruvate in the absence or presence of dichloroacetate. It is concluded that the inhibition by glucagon of pyruvate gluconeogenesis is not due to an inhibition of pyruvate dehydrogenase.

0162 THE ROLE OF METHIONINE IN GLUTATHIONE BIOSYNTHESIS BY ISOLATED HEPATOCYTES. (Eng.) Reed, D. J.; Orrenius, S. (Dept. Biochemistry and Biophysics, Oregon State Univ., Corvallis, OR 97331). *Biochem. Biophys. Res. Commun.* 77(4): 1257-1264; 1977.

The role of methionine and cysteine in glutathione synthesis was studied in rat hepatocytes after experimental *in vivo* depletion of liver glutathione. Hepatocytes freshly isolated from diethylmaleate (0.6 mmol/kg, i.p.)-treated rats were shown to perform net biosynthesis of intracellular glutathione at approximately an *in vivo* rate. ^{35}S from ^{35}S-cysteine or ^{35}S-methionine each contributed to the formation of glutathione to about the same extent, up to 75% and 61%, resp., depending upon the amino acid concentration in the medium. The sulfur atom of methionine makes a significant contribution to the synthesis of the thiol of glutathione, probably via the formation of cystathionine.

0163 HALOTHANE HEPATOTOXICITY: ENHANCEMENT BY POLYCHLORINATED BIPHENYL PRETREATMENT. (Eng.) Reynolds, E. S.; Moslen, M. T. (Univ. Texas Medical Branch, Galveston, TX 77550). *Anesthesiology* 47(1):19-27; 1977.

Halothane hepatotoxicity was studied in male Sprague-Dawley rats pretreated with polychlorinated biphenyl (Aroclor 1254, 150 μmol/kg, 7 days). Within 2 hr of the end of anesthesia (0.85% for 5 hr), the SGOT and SGPT levels were increased to above normal, and morphologic injury was recognized in the centrilobular parenchymal cells of the pretreated animals. Necrosis of the centrilobular hepatocytes was prominent within 24 hr. The ultrastructure of the hepatic tissue at intervals after anesthesia indicated that the injury primarily involved the endoplasmic reticulum (ER), progressing from dispersion of the rough ER to vacuolization of the rough ER and coalescence of the smooth compenents of this membranous system into tubular aggregates. These changes were preceded by selective deactivation of the mixed-function oxidase components, cytochrome P-450 and zoxazolamine hydroxylase, and early transient increases in conjugated dienes of lipids. Although pretreatment with Aroclor 1254 potentiated halothane hepatotoxicity, neither covalent binding of labeled halothane to liver proteins nor excretion of water-soluble labeled metabolites in the urine was enhanced 24 hr after the i.p. administration of a low nonhepatotoxic or high hepatotoxic dose of ^{14}C-1-labeled halothane (10 and 10,000 μmol/kg, resp.). These findings suggest that the toxic pathway(s) for the biotransformation of halothane do not involve increased production of water-soluble or protein-bound metabolites.

0164 INCREASE OF SERUM VERY LOW DENSITY LIPOPROTEINS IN RATS AFTER ADMINISTRATION OF α-HEXACHLOROCYCLOHEXANE. (Eng.) Grajewski, O.; Oberdisse, E. (Pharmakologisches Institut der Freien Universitat Berlin, Thielallee 69/73, D-1000 Berlin 33, Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 298(2):129-135; 1977.

The effect of α-hexachlorocyclohexane (α-HCH) on serum lipids and lipoproteins was studied in female Wistar rats. After enteral administration of 200 mg/kg α-HCH the rats developed hyperlipemia. Forty-eight hours after the administration, serum triglycerides were increased by 300%, whereas both serum cholesterol and serum total phospholipids only increased by about 45%. Serum free fatty acids were not significantly altered. Fractionation of the serum lipoproteins by ultracentrifugation showed that the hyperlipemia was due to a fivefold increase in serum very low density lipoproteins. Hepatic triglyceride secretion, calculated after i.v. injection of Triton WR 1339, was increased in animals pretreated with α-HCH. Corresponding to this observation, drugs known to diminish the triglyceride secretion of the liver, such as actinomycin D, cycloheximide, glucagon, orotic acid, CFT 1201, and CFT 1042 reduced the α-HCH-induced hyperlipemia. It is concluded that hyperlipoproteinemia after α-HCH is due to an increased hepatic very low density lipoprotein secretion. Blood sugar level was decreased in fasting animals after α-HCH treatment. Earlier experiments suggest that this effect is due to a decreased gluconeogenesis in the liver.

0165 TOXIC EFFECTS OF INDOCYANINE GREEN ON RAT LIVER MITOCHONDRIA. (Eng.) Laperche, Y.; Oudea, M. C.; Lostanlen, D. (Hopital Henri Mondor, 94010 Creteil, France). *Toxicol. Appl. Pharmacol.* 41(2):377-387; 1977.

Experiments were carried out to determine whether the decrease in bile flow seen in the rat after the administration of indocyanine green (ICG) at rates exceeding the maximal capacity of hepatic transport is dependent on ICG's effects on liver mitochondria. ICG (68-171 nmol/mg protein) inhibited the oxygen consumption of rat liver mitochondria *in vitro*. In state IV respiration, an acceleration of oxygen

consumption occurred prior to this inhibition. ICG induced mitochondrial swelling in an isotonic KCl and K^+ acetate medium but not in a 0.27 M sucrose medium. The increase in the ^{42}K diffusion space of isolated mitochondria was significantly larger than that of the ^{14}C sucrose diffusion space, indicating that ICG renders the mitochondrial membrane permeable to K^+ ions. At the same time, an efflux of H^+ ions from mitochondria was observed. The inhibition of mitochondrial oxygen consumption could be the result of a decrease in substrate penetration, as suggested by the diminution in NAD reduction by glutamate or isocitrate in the presence of ICG, without change in the activity of the corresponding dehydrogenases. A nigericin-like-mediated K^+ for H^+ antiport is proposed to account for the effects of ICG upon mitochondrial membrane permeability.

0166 EFFECTS OF ANESTHETIC AGENTS ON THE BILIARY EXCRETION OF IOPANOATE IN THE RAT. (Eng.) Cooke, W. J.; Cooke, L. (Univ. Massachusetts Medical Sch., 55 Lake Ave. North, Worcester, MA 01605). *Drug Metab. Dispos.* 5(4):377-385; 1977.

The effects of three anesthetics (ether, pentobarbital, and urethane) and decerebration on the metabolism and the biliary excretion following iopanoate administration were compared in Sprague-Dawley rats. Following the administration of ^{125}I-iopanoate glucuronide (13 μmol/kg, i.v.), its biliary excretion was 65% lower in animals anesthetized with ether than in those anesthetized with urethane (800 mg/kg, i.p.); when a high dose of iopanoate (169 μmol/kg) was administered, the difference was less marked but still significant ($p<0.01$). Biliary excretion of iopanoate glucuronide in animals anesthetized with pentobarbital (45 mg/kg, i.p.) was intermediate to that in animals anesthetized with ether and urethane. Decerebrate animals excreted iopanoate glucuronide at rates indistinguishable from those in animals anesthetized with urethane. The variations in biliary excretion of iopanoate glucuronide were accompanied by changes in the plasma decay of iopanoate. The positive relationship between the hepatic and plasma concentrations of iopanoate was unaffected by the anesthetic agent used. Ether significantly decreased the hepatic iopanoate glucuronide/iopanoate ratio, when compared with animals anesthetized with urethane or with decerebrate animals. The relation between the hepatic content of iopanoate glucuronide and the biliary excretion of iopanoate glucuronide was not affected by the anesthetic. The biliary excretory process appears to be saturable with an apparent V_{max} for iopanoate glucuronide of 4 μmol/min/kg and an apparent K_m of 1 μmol/g liver. These data indicate that the hepatic metabolism of iopanoate to iopanoate glucuronide is depressed by ether and by pentobarbital anesthesia in comparison to urethane anesthesia or decerebration.

0167 EFFECT OF ESSENTIAL AND NONESSENTIAL FATTY ACIDS IN COMPLEX MIXTURE ON FATTY ACID COMPOSITION OF LIVER LIPIDS. (Eng.) Caster, W. O.; Andrews, J. W., Jr.; Mohrhauer, H.; Holman, R. T. (Sch. Home Economics, Univ. Georgia, Athens, GA 30602). *J. Nutr.* 106(12):1809-1816; 1976.

The fatty acid composition of liver lipids was determined and was related to that of diet lipids fed to weanling rats for 60 days. The diet lipids constituted 5% of diet and were fed in the form of 24 different fatty acid ethyl ester mixtures containing 10 major fatty acids proportioned so that the intake of each one was poorly correlated with the intake of the remainder. The correlation coefficient between the amount of fatty acid in the diet and in the liver lipids was close to zero, except for linoleate and docosahexanoate acids (laurate, myristate, oleate). Linoleate fed in lipid mixtures was more effective in raising the concentration in liver lipids than previous studies had shown when the fatty acid was fed alone. The nutritional requirement for linoleate, an essential fatty acid, and the dose-response curve of liver concentration was similar when linoleate was fed in mixtures and alone. It is concluded that each of the polyunsaturated fatty acid components fed in a complex mixture behaves as expected from observations in experiments with diets containing lipids in the form of limited numbers of pure esters.

0168 DIFFERENTIAL EFFECTS OF DIETARY METHYL ESTERS OF LONG-CHAIN SATURATED AND POLYUNSATURATED FATTY ACIDS ON RAT LIVER AND ADIPOSE TISSUE LIPOGENESIS. (Eng.) Clarke, S. D.; Romsos, D. R.; Leveille, G. A. (Dept. Food Science and Human Nutrition, Michigan State Univ., East Lansing, MI 48824). *J. Nutr.* 107(7):1170-1181; 1977.

Four experiments were conducted to investigate the influence methyl esters of $C_{16:0}$, $C_{18:0}$, $C_{18:1}$, $C_{18:2}$, and $C_{18:3}$ fatty acids exert on rat liver and adipose tissue fatty acid synthesis and related enzymes when supplemented to a fat-free (FF) diet. A randomized complete block design, in which rats were matched for body weight and food intake, was used. Rats previously adapted to a meal-eating regimen (access to food from 0900-1200 hr) were fed a FF diet for 7 days prior to the addition of the respective dietary fatty acids. In experiments 1 to 3, all esters were supplemented at a level of 3% of the daily FF diet allotment. The apparent absorbabilities of $C_{16:0}$ and $C_{18:0}$ were 40% and 35%, resp., while the values for $C_{18:1}$, $C_{18:2}$, and $C_{18:3}$ were 88%, 87%, and 89%, resp. In comparison with the FF dietary treatment, polyunsaturated fatty acids ($C_{18:2}$ and $C_{18:3}$) reduced the rate of hepatic fatty acid synthesis and the activities of hepatic fatty acid synthetase (FAS) and malic enzyme (ME) in three of four experiments. In all four experiments, glucose-6-phosphate dehydrogenase (G6PD) activity was significantly depressed by $C_{18:2}$ and $C_{18:3}$. $C_{18:1}$ produced an intermediate rate of *in vitro* liver fatty acid synthesis, but exerted no significant effect on hepatic lipogenic enzyme activities. Adding $C_{16:0}$ or $C_{18:0}$ to the FF diet had no depressive effect on hepatic fatty acid synthesis, but $C_{18:0}$ supplementation was associated

with a significant increase in hepatic FAS, G6PD, and ME activities. Neither unsaturated nor saturated fatty acids affected adipose tissue FAS, G6PD. The lack of influence of $C_{16:0}$ and $C_{18:0}$ may have been partially due to their poor absorbability. Therefore, a level of 7% $C_{16:0}$ supplementation was compared with 3% $C_{18:2}$ or $C_{18:3}$. This resulted in comparable amounts of fatty acid absorbed. Both $C_{18:2}$ and $C_{18:3}$ addition to the FF diet were equally effective in significantly depressing *in vivo* hepatic fatty acid synthesis, and in decreasing FAS, G6PD, and ME activities by more than 50%. $C_{16:0}$ had no suppressive action on liver fatty acid synthesis or related enzyme activities. Adipose tissue lipogenesis remained unchanged by dietary fat treatment. These data demonstrate that $C_{18:2}$ and $C_{18:3}$ specifically inhibit rat liver fatty acid synthesis, independent of carbohydrate intake. Under these conditions adipose tissue lipogenesis was unaffected by low levels of dietary fat, regardless of the degree of unsaturation.

0169 THE EFFECT OF DIETARY LIPID PEROXIDES, STEROLS AND OXIDISED STEROLS ON CYTOCHROME P450 AND OXIDATIVE DEMETHYLATION IN THE ENDOPLASMIC RETICULUM. (Eng.) Lambert, L.; Wills, E. D. (St. Bartholomew's Hosp., Charterhouse Square, London ECIM 6BQ, England). *Biochem. Pharmacol.* 26(15):1417-1421; 1977.

The effects of dietary linoleate, oxidized linoleate, lipid peroxides, cholesterol, cholesterol hydroperoxides, and squalene on the concentration of cytochrome P450 and rate of oxidative demethylation were examined in the endoplasmic reticulum of the rat liver. Addition of 5% methyl linoleate to a diet containing 5% lard caused a large increase in the rate of oxidative demethylation. Oxidized methyl linoleate (5%) was much less effective and also depressed the rate of oxidation when added to a diet containing corn oil. A supply of dietary linoleic acid or other polyunsaturated fatty acids is essential for maximum rate of oxidative demethylation in the endoplasmic reticulum and for maximum induction by phenobarbitone. Dietary lipid peroxide did not increase the rate of oxidative metabolism in the endoplasmic reticulum or its induction. Feeding a diet that included 10% herring oil and that contained only 1.7% linoleic acid caused a very rapid rate of oxidative demethylation and a high concentration of cytochrome P450 in the liver endoplasmic reticulum. Induction by phenobarbitone was also very effective after feeding this diet. The unsaponifiable fraction of herring oil, added to the lard diet, effectively increased the rate of oxidative demethylation to a value similar to that observed after feeding a diet containing 10% herring oil. The addition of cholesterol (2 g/kg diet) or of squaline (1 g/kg diet) to diets containing 10% lard were less effective than the unsaponifiable fraction of herring oil, but caused a large increase in the content of cytochrome P450 and rate of oxidative demethylation in the endoplasmic reticulum. The induction of phenobarbitone was also enhanced. Cholesterol hydroperoxide was no more effective than cholesterol. A supply of dietary sterols is essential for maximum activity of oxidative demethylation in the liver endoplasmic reticulum, especially when linoleic acid is in restricted supply, and for maximum induction by phenobarbitone. The nonsaponifiable fraction of herring oil cannot be completely replaced in the diet by pure cholesterol, cholesterol hydroperoxide, or squalene.

0170 LIVER AND STEROID HORMONES. II. INFLUENCING THE HEPATIC METABOLISM BY STEROID HORMONES. (Ger.) Kley, H. K.; Kruskemper, H. L. (2. Medizinische Klinik, Moorenstrasse 5, D-4000 Dusseldorf 1, W. Germany). *Med. Klin.* 72(7): 231-239; 1977.

Studies on relationships between steroid hormones and the liver are reviewed. Cortisol was found to induce the activity of such hepatic enzymes as tryptophan pyrrolase, tyrosine transaminase, pyruvate-glutamate transaminase, pyruvate carboxylase, phosphoenolpyruvate carboxykinase, fructose-1,6-diphosphatase, glucose-6-phosphatase, and glucogen synthetase. No glycogen synthesizing enzymes were found in the liver of adrenalectomized, fasting animals. Twenty percent of patients with Cushing's syndrome have an increased blood glucose level, and 90% show pathological blood sugar load tests. Estrogens increase the hepatic DNA and RNA levels and increase and modify the activity of hepatic enzymes, such as glucose-6-phosphate dehydrogenase and delta-aminolevulinic acid synthetase. Estrogens increase the liver glycogen level, the plasma insulin, total lipid, triglyceride, free fatty acid, cholesterol, and lipoprotein concentrations, and the glycolytic capacity of the liver. The concentrations of practically all specific hormone-binding proteins are increased by estrogens and gestagens, and reduced by androgens. Testosterone increases the activity of fumarate hydratase, transaminase, and delta-amino acid oxidase.

0171 EFFECTS OF CONTRACEPTIVE STEROIDS AND PREGNANCY ON BILE COMPOSITION AND KINETICS IN THE BABOON. (Eng.) Morrissey, K.; Panveliwalla, D.; McSherry, C.; Deitrick, J.; Niemann, W.; Gupta, G. (New York Hosp.-Cornell Medical Center, New York, NY 10021). *J. Surg. Res.* 22(6):598-604; 1977.

The effect of the contraceptive steroids, ethinylestradiol (EE) and Norethindrone (NET), and of pregnancy on bile acid metabolism was studied in the baboon, an animal that spontaneously forms predominantly cholesterol gallstones as does man. The contraceptive steroids were administered continuously (EE, 65 µg/day; NET, 100 µg/day) via solid Silastic pellets implanted s.c. in the abdomen or sternal region of five baboons (1 male) according to the following protocol: EE, 8 weeks; EE+NET, 10 weeks; NET, 8 weeks. Seven female baboons were used for the pregnancy studies performed prior to and after pregnancy as well as during the second and third trimesters. Gallbladder bile was aspirated percutaneously after a 16-hr fast and on alternate days after i.v. injection of isotopically-labeled bile acids. The re-

sponse to synthetic sex hormones and to pregnancy was similar: a significant ($p<0.01$-0.05) decrease occurred in biliary cholesterol level and in chenodeoxycholic acid pool size and synthesis rate. When pregnancy had ended or the estrogen component of the contraceptive steroids (EE) was removed, these values reverted to control or antepartum levels. As a result of the concomitant increase in deoxycholic acid pool size, there was no net change in total bile acid pool size during any of the periods of administration of EE or EE+NET, but there was a significant ($p<0.05$) decrease during the third trimester of pregnancy. Over the past 5 yr, a 6% incidence of cholelithiasis among 219 female baboons has been observed, but none occurred in the present study.

0172 INFLUENCE OF DIETARY FAT ON FATTY LIVERS OF CHOLINE-DEFICIENT RATS. (Eng.) Carroll, C.; Williams, L. (Agricultural Experiment Station, Univ. Arkansas, Fayetteville, AR 72701). *J. Nutr.* 107(7):1263-1268; 1977.

The influence of the type of dietary fat on the nature and severity of liver lipid changes induced by low choline diets was investigated in male weanling rats. Beef tallow, a blend of tallow and safflower oil, or safflower oil (SO) each provided 48% of total energy value of a control diet (0.20 g choline chloride/100 g diet) and a low choline diet (no added choline). Livers from choline-deficient rats fed tallow, blend, or SO diets contained approximately 4.5, 5, and 2.5 times as much lipid, resp., as livers from corresponding control groups. Liver lipids from rats fed SO diets, as compared with those from rats fed tallow diets, contained lower percentages of saturated and mono-unsaturated fatty acids, and higher percentages of polyunsaturated fatty acids. Values for groups fed blend diets were intermediate, except for 18:0 and 20:4 in the control group. Choline deficiency resulted in significant increases in proportions of the predominant fatty acid(s) of the dietary fat in liver lipids (but not in fat pad lipids) at the expense of 18:0 and 20:4. Ratios of 16:0 to 18:0 in both liver and fat pad lipids were greater in choline-deficient rats than in corresponding control groups, probably reflecting greater fatty acid synthesis.

0173 REVERSAL OF OROTIC ACID-INDUCED FATTY LIVER IN RATS BY CLOFIBRATE. (Eng.) Novikoff, P. M.; Edelstein, D. (Albert Einstein Coll. Medicine, 1300 Morris Park Ave., Bronx, NY 10461). *Lab. Invest.* 36(2):215-231; 1977.

Morphologic observations and biochemical and immunoelectrophoretic results of the reversal of orotic acid-induced fatty liver by clofibrate (CPIB) are presented. The addition of 1% orotic acid to a sucrose-enriched semipurified diet resulted in markedly fatty liver when fed to rats for 7-22 days. Light microscopy revealed lipid droplets distributed throughout the cytoplasm of all hepatocytes. Electron microscopy showed that all the endoplasmic reticulum (ER) was broken into vesicles. Within the interior (cisterna) of each vesicle, one or more lipid droplets were present. Morphologic signs of normal lipid transport (ER to Golgi apparatus to space of Disse) disappeared. The Golgi elements were flattened and lacked very low density lipoprotein particles; the Golgi-derived secretory vacuoles were not present. Biochemical analyses showed an increase in hepatic triacylglycerol levels, to approximately 8 times the levels of sucrose-fed controls by the 7th day, 18 times by the 15th day, and 25 times by the 22nd day. Hepatic cholesterol levels increased two- to fourfold. Serum triacylglycerol levels fell markedly, and serum cholesterol levels were reduced. The apoprotein B component of plasma lipoproteins was practically absent at 7 days and increased slightly at 22 days. By 8-16 days after adding CPIB (0.25%) to the diet, the ER of the hepatocytes returned to its usual parallel configuration, and lipid droplets were not seen within its cisternae. Morphologic features of normal lipid transport reappeared. Lipid-containing residual bodies were common. Peroxisomes were more numerous than in hepatocytes of control rats. Liver triacylglycerol levels approached sucrose-fed control levels, and serum triacylglycerol levels returned to chow-fed control levels. Hepatic cholesterol levels were similar to those of sucrose-fed and chow-fed controls, whereas serum cholesterol levels were lower. Serum apoprotein B levels returned to chow-fed control levels. A sequence of events terminating in the removal of lipid from the hepatocytes is suggested by observation of morphologic changes following CPIB administration.

0174 THE PHARMACOKINETIC PATTERN OF PENTOBARBITAL IN HEPATIC DAMAGE. (Ger.) Ossenberg, F. W. (I. Med. Abt., Allg. Krankenhaus Barmbek, Rubenkamp 148, 2000 Hamburg 60, W. Germany). *Z. Gastroenterol.* 15(6):393-403; 1977.

The kinetic pattern of distribution and elimination of pentobarbital (PB) was tested in rats with hepatic damage and in 21 patients with liver cirrhosis. After i.v. injection of PB in normal rats, the PB blood level curve showed a dual exponential decreasing course. After total hepatectomy, an initial decrease of PB blood level occurred, corresponding to the distribution of the drug in the body. Thereafter, the PB blood level remained constant, since PB was no longer eliminated due to lack of a liver. Microsomal PB metabolizing liver enzymes and the rate of elimination of PB were reduced in rats after portacaval anastomosis alone or by arterialization of the liver (end-to-end anastomosis between the renal artery and the stump of the portal vein); the cytochrome P450 content of the liver and PB clearance were significantly reduced. Presumably, a hepatotrophic factor in the portal vein blood is necessary for the normal functioning of the hepatic microsomal enzyme system. Rats with extrahepatic cholestasis induced by ligation of the bile ducts had a significantly reduced PB clearance and a decreased initial volume of distribution. PB elimination and initial volume of distribution were significantly

reduced in some of the 21 patients with liver cirrhosis; the cause of this disorder has not yet been elucidated. When the rate of PB metabolism is reduced, a delayed action of the drug must be expected. Repeated administration may lead to accumulation in the body.

0175 INDUCTION OF HEPATIC MONO-OXYGENASE SYSTEMS OF PREGNANT RATS WITH PHENOBARBITAL AND 3-METHYLCHOLANTHRENE. (Eng.) Guenthner, T. M.; Mannering, G. J. (Univ. Minnesota Medical Sch., Minneapolis, MN 55455). *Biochem. Pharmacol.* 26(1): 577-584; 1977.

The selective mechanism governing induction of hepatic mono-oxygenase systems in adult rats during pregnancy was studied. Rats were injected i.p. daily for 4 days with sodium phenobarbital (40 mg/kg in saline), 3-methylcholanthrene (16 mg/kg in corn oil), or progesterone (50 mg/kg in corn oil) in a volume of 2 ml/kg. Ethylmorphine *N*-demethylase, a cytochrome P-450-dependent system, was induced about fourfold in nonpregnant rats but only twofold in pregnant rats when phenobarbital was administered alone. Also the induction of microsomal cytochrome P-450 was about half that observed with nonpregnant rats ($p<0.05$). This impairment of phenobarbital induction occurred within 3 days of conception and disappeared within 5 days after parturition. 3-Methylcholanthrene given alone produced no increase in ethylmorphine metabolism. The depressed response of the maternal liver to phenobarbital induction was partially reversed by the co-administration of 3-methylcholanthrene. The administration of a higher dose (80 mg/kg/day for 4 days) of sodium phenobarbital (PB 80) overcame the pregnancy-related lowered response to phenobarbital induction observed with smaller dose (40 mg/kg/day for 4 days) of the barbiturate (PB 40). Ethylmorphine demethylation in moles HCHO/g/liver/hr was 5.12 ± 0.7 in PB 40 pregnant rats and 11.32 ± 1.2 in PB 80 pregnant rats, which compared to the nonpregnant rats PB 40 value of 11.3 ± 1.4. Progesterone (50 mg/kg) lowered the induction of cytochrome P-450 ($p<0.05$), but induction of mono-oxygenase activity was not lowered. The similarity in responses of the maternal and fetal livers to inducing agents suggests that a common regulatory mechanism operates both in the fetus and in pregnant female rats.

0176 EFFECT OF HYPOPHYSECTOMY ON THE STIMULATION OF LIVER GROWTH BY α-HEXACHLOROCYCLOHEXANE, PHENOBARBITAL, AND PARTIAL HEPATECTOMY IN THE RAT. (Eng.) Schulte-Hermann, R.; Landgraf, H.; Koransky, W. (Institut fur Toxikologie und Pharmakologie der Philipps-Universitat Marburg, Pilgrimstein 2, D-3550 Marburg a.d. Lahn, W. Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 298(2):137-142; 1977.

Studies were carried out to determine whether the hypophysis or its hormones are involved in the stimulation of liver cell proliferation elicited by α-hexachlorocyclohexane (α-HCH), phenobarbital (PB), or partial hepatectomy. In hypophysectomized CFHB rats, α-HCH (200 mg/kg, 15-17 days after hypophysectomy and 100 mg/kg 4, 7, and 8 days later) and PB (80 mg/kg on 4 consecutive days from day 18-20 after hypophysectomy and 100 mg/kg on the following 4 days) induced an increase in liver mass, but no increase in liver DNA. Hypophysectomy without additional treatment resulted in a decrease of liver size and RNA, while the DNA content remained unchanged, thereby leading to a relative DNA surplus. One-third hepatectomy in hypophysectomized animals led to a small increase of hepatic DNA only; after two-thirds hepatectomy, 75-80% of the original liver DNA was restored. In rats with an intact hypophysis, losses of liver DNA are known to be restored completely. The findings suggest that the relative DNA surplus in hypophysectomized rats prevents the stimulation of DNA synthesis by weak growth stimuli such as α-HCH, PB, and one-third hepatectomy. If the relative DNA surplus is eliminated by partial hepatectomy, the inducers do produce DNA multiplication. It is concluded that the induction of liver growth and cell multiplication by α-HCH and PB does not require the presence of the hypophysis or one of its hormones.

0177 INDUCTION OF HEPATIC MONO-OXYGENASE SYSTEMS IN FETAL AND NEONATAL RATS WITH PHENOBARBITAL, POLYCLINIC HYDROCARBONS AND OTHER XENOBIOTICS. (Eng.) Guenthner, T. M.; Mannering, G. J. (Natl. Inst. Child Health and Human Development, NIH, Bethesda, MD 20014). *Biochem. Pharmacol.* 26(7):567-575; 1977.

The hepatic P-450 hemoprotein-dependent mono-oxygenase systems were studied in fetal and neonatal Sprague-Dawley rats born to mothers injected throughout pregnancy with a number of exogenous agents (sodium phenobarbital, 3-methylcholanthrene, dieldrin, chlordane, DDT, 2,4,5-trichlorophenoxyacetic acid [2,4,5-T], α- and β-naphthoflavone, benzo[a]pyrene, dibenz[a,c]anthracene, 3β-hydroxy-20-oxopreg-5-ene-16α-carbonitrile [PCN], and 2-diethylaminoethyl-2,2-diphenylvalerate [SKF 525-A]). Microsomal preparations were made from livers collected on days 13, 16, and 18-22 postconception and 23-25 days postpartum. Phenobarbital (total dose, 80 mg/kg) did not induce the cytochrome P-450-dependent mono-oxygenase activities, aminopyrine or ethylmorphine *N*-demethylase in the fetal liver. Benzo(a)pyrene hydroxylase activity, a cytochrome P_1-450-dependent mono-oxygenase activity, was induced in fetal liver by 3-methylcholanthrene (16 mg/kg). All three enzymes were active in neonatal liver. When 3-methylcholanthrene, SKF 525-A, PCN, or dibenz(a,c)-anthracene were administered with phenobarbital, the apparent suppression of phenobarbital-induced mono-oxygenase activities in the fetal liver was partially reversed. The other inducing agents tested were inactive in reversing the suppression. It is concluded that a control mechanism operates in the fetal rat that selectively suppresses induction of cytochrome P-450 but not P_1-450 synthesis. Certain pharmacological agents can reverse this suppression.

0178 LONG-TERM STUDIES ON CHEMICALLY INDUCED LIVER ENLARGEMENT IN THE RAT. I. SUSTAINED INDUCTION OF MICROSOMAL ENZYMES WITH ABSENCE OF LIVER DAMAGE ON FEEDING PHENOBARBITONE OR BUTYLATED HYDROXYTOLUENE. (Eng.) Crampton, R. F.; Gray, T. J. B.; Grasso, P.; Parke, D. V. (British Industrial Biological Res. Assoc., Woodmansterne Rd., Carshalton, Surrey, England). *Toxicology* 7(3):289-306; 1977.

As part of a study on the relationships between liver enlargement, induction of drug metabolizing enzymes, and certain forms of liver damage, sequential biochemical and morphological observations were made on the livers of female Wistar rats fed diets containing 0.4% butylated hydroxytoluene (BHT) or 0.25% phenobarbitone for up to 80 weeks. Both compounds increased the relative liver weight by 35% and 34% with BHT and phenobarbitone, resp., and produced marked enhancement of the activities of ethylmorphine *N*-demethylase, aniline 4-hydroxylase, biphenyl 4-hydroxylase, and NADPH-cytochrome c reductase and of the contents of cytochromes P-450 and b_5 and microsomal protein (by 95%, 15%, 91%, 32%, 83%, 78%, and 50%, resp.). These changes were evident after 1 week of treatment and persisted until treatment was stopped after 80 weeks. The only morphological changes observed throughout the treatment period were centrilobular cell enlargement and hypertrophy of the smooth endoplasmic reticulum. Histochemical examination revealed a centrilobular depression of glucose-6-phosphatase, but there was no disturbance of the normal pericanalicular distribution of lysosomes. All changes observed with either compound were reversible on cessation of treatment at week 80. These results support the concept that liver enlargement accompanied by induction of drug metabolizing enzymes represents an adaptive response, and they provide a basis for the interpretation of pathological changes developing in the enlarged liver unaccompanied by drug metabolizing enzyme induction.

0179 LONG-TERM STUDIES ON CHEMICALLY INDUCED LIVER ENLARGEMENT IN THE RAT. II. TRANSIENT INDUCTION OF MICROSOMAL ENZYMES LEADING TO LIVER DAMAGE AND NODULAR HYPERPLASIA PRODUCED BY SAFROLE AND PONCEAU MX. (Eng.) Crampton, R. F.; Gray, T. J. B.; Grasso, P.; Parke, D. V. (British Industrial Biological Res. Assoc., Woodmansterne Rd., Carshalton, Surrey, England). *Toxicology* 7(3):307-326; 1977.

A long-term study with Safrole and Ponceau MX was carried out in female Wistar rats to determine whether changes in hepatic drug metabolizing enzyme activity are correlated with the development of liver injury. Sequential biochemical and morphological observations were made on the livers of rats fed a diet containing 0.25% safrole or 1.0% Ponceau MX for up to 85 weeks. After the administration of safrole for 7 days, the relative liver weight was increased by 20% above the control value, and the activity of the drug metabolizing enzymes was enhanced by 75-210%. This enzyme induction was short-lived, however, and continued administration of safrole led to inhibition of drug metabolizing activity. Despite this, the liver remained enlarged, and concentrations of NADPH-cytochrome c reductase, cytochrome b_5, and microsomal protein were markedly elevated. During the initial phase of enzyme induction, no histochemical or morphological abnormalities were evident, but the loss of this inductive response was associated with histochemical and ultrastructural evidence of liver damage. The enlarged liver produced by Ponceau MX was initially accompanied by moderate induction of the drug metabolizing enzymes, followed by a gradual reversal as treatment continued. Histochemical evidence of liver damage was encountered after only 1 week of treatment. The early cytological changes produced by both compounds were followed by the development of fatty change, cell necrosis and, ultimately, hepatic nodules. Liver enlargement unaccompanied by induction of the drug metabolizing enzymes may provide an early index of hepatotoxicity.

0180 SYNTHESIS OF GLYCOPROTEINS IN THE GOLGI COMPLEX OF THE MOUSE GALLBLADDER EPITHELIUM DURING FASTING, REFEEDING, AND GALLSTONE FORMATION: A LIGHT MICROSCOPIC AUTORADIOGRAPHIC AND QUANTITATIVE ELECTRON MICROSCOPIC STUDY. (Eng.) Wahlin, T. (Dept. Histology and Pathology, Univ. Umea, S-901 87 Umea, Sweden). *Histochemistry* 51(2/3): 133-140; 1977.

The mouse gallbladder epithelium was studied during fasting, refeeding, and experimental gallstone formation to establish if quantitative and qualitative variations in food intake in mice are reflected in the incorporation pattern of radioisotope into epithelial cell glycoproteins and in morphological changes at the site of glycoprotein synthesis, i.e., in the Golgi complexes of the gallbladder principal cells. Control gallbladder epithelium, examined after an intraperitoneal injection of [^{3}H]galactose (0.1 mCi) showed labeling of the supranuclear Golgi region in all normal principal cells at 10 min and an increased radioactivity throughout the apical cytoplasm at 25 min. At 40 min, after [^{3}H]galactose injection, silver grains were mainly present at the cell surface. Autoradiographs processed 25 min after [^{3}H]galactose injection that followed a 48-hr fast showed decreased supranuclear and apical radioactivity. After refeeding (12 hr), there was an enhanced activity in both these regions, roughly equal to the activity observed in controls. Animals fed a lithogenic diet for one month showed a marked increase of radioactivity, mainly in cells of crypts and invaginations of the gallbladder mucosa. Morphometric measurements of the Golgi apparatus revealed that deprivation of food reduced to 2.7% ($p<0.01$) and refeeding restored (to 4.2%, $p<0.05$) the volume density of the Golgi apparatus. In the course of gallstone formation there was a further increase to 6.4% ($p<0.001$) in the volume density of the Golgi complexes as compared to that in controls (4.4%). It is concluded that the synthesis of glycoproteins in the mouse gallbladder principal cells is dependent both on the nutritional

state of the mice and the qualitative composition of the diet given to the animals.

0181 ENDOGENOUS INHIBITORS OF PROTEIN SYNTHESIS IN ISCHAEMIC LIVERS. (Eng.) Bernelli-Zazzera, A. (Istituto Patologia Generale, Via Mangiagalli 31, 30133 Milano, Italy). *Panminerva Med.* 18(9/10):326-331; 1976.

Factors leading to the conclusion that the inhibition in protein synthesis seen in the ischemic liver is due to endogenous inhibition are reviewed. The interruption of the blood supply impairs protein synthesis in various cellular or cell-free preparations from ischemic livers (tissue slices, post-mitochondrial supernatants, microsomes, isolated polysomes, cell sap). This inhibition depends on the decay of many metabolic reactions and structures involved in the pathway of protein synthesis. Ribosomal RNA decreases; polysomes break down; there is an actual drop in the number of active particles, rather than a proportional loss of functional capacity of all the ribosomes; and ribosome-membrane relationships become looser. In the cell sap, the activity of both synthetases and transferases is reduced. In ischemic cells, the energy to drive the reaction of protein synthesis is drastically lowered, due to mitochondrial damage. Initiation of new peptides seems to be mainly inhibited, but there is also evidence of impairment of elongation of the initiated chains. At least some of the changes in protein synthesis are related to the increase in concentration of endogenous metabolites, such as ADP, AMP, NADH, and other unidentified molecules accumulating in ischemic liver cells, and possibly to changes in the ratios that describe the "energy state" and the "redox state" of the liver cell.

0182 COMPARATIVE STUDY OF PROTOPORPHYRINS IN ERYTHROPOIETIC PROTOPORPHYRIA AND GRISEOFULVIN-INDUCED MURINE PROTOPORPHYRIA. BINDING AFFINITIES, DISTRIBUTION, AND FLUORESCENCE SPECTRA IN VARIOUS BLOOD FRACTIONS. (Eng.) Poh-Fitzpatrick, M. B.; Lamola, A. A.; Zalar, G. L.; Weinstein, M.; Doleiden, F.; Freeman, M. (Columbia Univ. Coll. Physicians and Surgeons, New York, NY 10032). *J. Clin. Invest.* 60(2):380-389; 1977.

The use of griseofulvin-induced murine protoporphyria (GFPP) as a model for erythropoietic protoporphyria (EPP) and the use of tissue localization of protoporphyrin(s) (PP) as a criterion for the tissue source of PP were assessed in Sprague-Dawley mice. Stroma from GFPP mice and EPP patients contained only a small fraction (0.5%) of the total RBC PP. Thus, 99.5% of the PP was associated with the cytosol hemoglobin (Hb) in both cases. The close agreement in PP distribution among the plasma, stroma, and Hb pools failed to help define any difference in sites of PP overproduction in GFPP and EPP. However, as shown by fractionation of RBC into populations of progressive age on discontinuous density gradients, PP distributions among RBC of different ages are diametrically opposed in GFPP and EPP. The murine RBC became more PP-laden with age in circulation, but there was a rapid loss of PP with age in EPP. Direct competition experiments showed that PP binds about 40 times more strongly to human Hb than it does to human serum albumin (HSA). The molar ratio of Hb to HSA is about 6:1. Assuming that all other plasma sites contribute to PP binding as importantly as HSA, the ratio of PP bound to Hb:PP bound to plasma proteins at equilibrium can be expected to be at least 120:1. This strong affinity can provide the driving force necessary for the net diffusion of PP from the plasma to the intracellular Hb in GFPP mice and it can explain the buildup of the high RBC PP levels. The opposite rapid efflux of intra-RBC PP into plasma occurs despite this strong Hb affinity, implying continuous, efficient clearance of PP from the plasma by the liver. These and other data suggest that liver-formed PP cannot significantly contribute to the circulating PP pool.

0183 THE HEPATIC HANDLING OF ^{131}I-LABELED SULFOBROMOPHTHALEIN IN THE DOG. COMPARISON WITH SULFOBROMOPHTHALEIN. (Eng.) Glasinovic, J. C.; Delage, Y.; Erlinger, S. (Hopital Beaujon, 100, Boulevard du General Leclerc, 92110 Clichy, France). *Biomedicine [Express]* 25(7):255-258; 1976.

The influence of added radioiodine on the hepatic extraction and biliary excretion of BSP was studied in dogs. Two types of experiments were performed. In the first, a compartmental analysis of BSP and ^{131}I-labeled BSP disappearance curves was performed after the simultaneous injection of BSP (5 mg/kg) and ^{131}I-labeled BSP (10 µCi). Both the plasma to liver and liver to bile transfer rates were significantly lower for ^{131}I-labeled BSP than for BSP; the liver to plasma transfer rates were not significantly different. In the second series of experiments, the hepatic uptake of BSP and ^{131}I-labeled BSP was estimated by the multiple indicator dilution technique. The extraction of BSP (59 ± 5%) was significantly higher than that of ^{131}I-labeled BSP (35 ± 5%, $p<0.001$). The labeling of BSP with ^{131}I significantly modifies the hepatic handling of the dye; ^{131}I-labeled BSP thus cannot be used as a tracer of BSP.

0184 HEPATIC UPTAKE AND BILIARY EXCRETION OF DIBROMOSULPHTHALEIN. ALBUMIN DEPENDENCE, INFLUENCE OF PHENOBARBITAL AND NAFENOPIN PRETREATMENT AND THE ROLE OF Y AND Z PROTEIN. (Eng.) Meijer, D. K. F.; Vonk, R. J.; Keulemans, K.; Weitering, J. G. (Bloemsingel 1, Groningen, Netherlands). *J. Pharmacol. Exp. Ther.* 202(1): 8-21; 1977.

The hepatic uptake and biliary excretion of 3,6-bromphenolphthalein-disulfonate (DBSP) in perfusion studies was compared with *in vivo* DBSP kinetics, and an attempt was made to establish a possible role of Y (ligandin) and Z proteins in the hepatic

uptake, storage, and excretion of this anion. The uptake and excretion of DBSP was studied in isolated perfused rat livers from control rats and from rats pretreated with phenobarbital (90 mg/kg, for 6 days) or with nafenopin (200 mg/kg, for 2 days). The flow rates of perfusate in control and treated groups were kept equal. Hepatic content of Y and Z proteins was determined by dye binding using gel filtration. DBSP was found to be associated with both Y and Z proteins in liver cytosol. DBSP clearance was studied using various concentrations of bovine albumin in the perfusion medium. Clearance was monoexponential with 3.0% albumin and under this condition mainly reflects biliary excretion of DBSP. At concentrations of 1.0%, 0.5%, and 0.2% albumin, a distinct biexponential plasma disappearance curve was found, from which rate constants for transfer from plasma to liver (K_{12}), liver to plasma (K_{21}), and liver to bile (K_{23}) were calculated. K_{12} was enhanced and K_{21} was lowered with decreasing albumin concentrations, whereas K_{23} was not affected. Phenobarbital treatment increased Y protein concentration twofold and left Z protein unchanged; nafenopin pretreatment slightly elevated Y protein and increased Z protein concentration threefold. With perfusate with 1% albumin, K_{12} was unchanged and K_{23} was increased in the phenobarbital experiments, whereas K_{21} was reduced. In the livers from nafenopin-pretreated rats, K_{21} was reduced and K_{12} and K_{23} were unchanged. Data on binding of DBSP at various albumin concentrations indicate that primary hepatic uptake (K_{12}) and reflux (K_{21}) both are influenced by variations in plasma albumin concentration but are not directly related to the free drug concentration. Phenobarbital pretreatment does not lead to an acceleration of primary hepatic uptake, a result possibly related to the flow-standardized condition compared with the *in vivo* situation. Induction of Y and Z proteins possibly contributes to the lower reflux of DBSP from liver into plasma, an effect that was also observed in the rat *in vivo*.

0185 CHANGES IN RENAL FUNCTION AND WATER-SALT METABOLISM IN RATS DURING INDUCTION OF TOXIC LIVER CIRRHOSIS. (Rus.) Gapon, L. I. (Chernovtsy Medical Inst., Chernovtsy, USSR). *Patol. Fiziol. Eksp. Ter.* (3):82-84; 1977.

0186 PECULIARITIES ASSOCIATED WITH THE DEVELOPMENT OF EXPERIMENTAL HEPATOCHOLECYSTITIS IN SUCKLING RABBITS. (Rus.) Tarakhovsky, M. L.; Lukyanova, E. M.; Emaikina, V. P.; Denisova, M. F.; Oleshko, L. D. (Kiev Scientific Res. Inst. Pediatrics, Obstetrics, Gynecology, Kiev, USSR). *Patol. Fiziol. Eksp. Ter.* (3):72-74; 1977.

0187 EFFECTS OF L-ARGININE HYDROCHLORIDE ADMINISTRATION ON THE DISTURBANCES OF LIVER GLUTAMINE METABOLISM FOLLOWING ACUTE AMMONIA INTOXICATION IN THE RAT. (Fre.) Petit, M. A.; Nordmann, J.; Nordmann, R. (INSERM, U-72, Hopital de la Salpetriere, 47, Boulevard de l'Hopital, F-75013 Paris, France). *Arch. Int. Physiol. Biochim.* 85(2):367-375; 1977.

0188 FREE FATTY ACIDS AND CHOLESTEROL AS POSSIBLE PARTICIPANTS IN REACTIONS OF FREE RADICAL OXIDATION OF LIPIDS IN ANIMAL TISSUES. (Rus.) Terekhova, S. F.; Burlakova, E. B.; Elizarova, T. I. (Inst. Chemical Physics, Acad. Sciences USSR, Moscow, USSR). *Vopr. Med. Khim.* 23(3):375-381; 1977.

0189 EFFECT OF LOCAL CARBAMATE ANESTHETICS ON MORPHOLOGICAL STRUCTURES OF THE LIVER AND SOME ENZYMES IN VIVO. (Cze.) Foltinova, J.; Helia, O. (Faculty Medicine, Univ. Komenskeho, Sasinkova 4, 884 24 Bratislava, Czechoslovakia). *Biologia* 32(3):195-207; 1977.

0190 SIGNIFICANCE OF THE HOUR-LONG SECRETION METHOD OF LIVER BILE DETERMINATION. (Rus.) Maximov, V. A.; Galkin, V. A. (No affiliation given). *Vrach. Delo* (2):107-109; 1977.

0191 RESISTANCE OF VARIOUS FORMS OF CYTOCHROME P-450 OF RAT LIVER TO THE ACTIONS DESTABILIZING THE MICROSOMAL MEMBRANE. (Rus.) Gromova, O. A.; Zakharova, N. E.; Lyakhovich, V. V.; Tsyrlov, I. B. (Inst. Clinical and Experimental Medicine, Siberian Branch USSR Acad. Medical Sciences, Novosibirsk, USSR). *Biull. Eksp. Biol. Med.* 83(5):553-555; 1977.

0192 FEATURES PECULIAR TO THE ACTION OF SODIUM AUROTHIOMALATE IN ACUTE TOXIC HEPATITIS. (Rus.) Korolenko, T. A.; Shkurupy, V. A.; Subbotin, V. M.; Yakobson, G. S. (Novosibirsk Medical Inst., Novosibirsk, USSR). *Biull. Eksp. Biol. Med.* 83(5): 539-542; 1977.

0193 EFFECT OF LOWERING OF PROTEIN LEVEL IN THE DIET ON THE TOXICITY OF BUTYLHYDROXYTOLUENE (BHT) IN THE RAT: PART II. ACTIVITY OF SELECTED ENZYMES IN A SUBACUTE EXPERIMENT. (Pol.) Nikonorow, M.; Karlowski, K. (Zakladu Badania Zywnosci i Przedmiotow Uzytku Panstwowego Zakladu Higieny, Warsaw, Poland). *Panstw. Zakl. Hig.* 28(3):225-236; 1977.

0194 HEXOBARBITAL-OXIDATION IN VIVO AND IN VITRO IN RATS WITH BILE DUCT LIGATION OR ANIT-CHOLESTASIS. (Ger.) Brachtel, D.; Gallenkamp, H.; Richter, E. (Medizinischen Universitatsklinik, Josef-Schneider-Str. 2, 8700 Wurzburg, W. Germany). *Z. Gastroenterol.* 15(6):378-380; 1977.

0195 TUMORIGENESIS FOLLOWING EXPERIMENTAL LITHIASIC CHOLECYSTITIS, WITH OR WITHOUT ADDED CARCINOGENS, IN MICE. (Fre.) Besancon, F.; Marche, C.; Habar, J. L.; Parrot, J.; Damianakis, N. (Clinique et Semiologie Medicales, Hotel-Dieu, Parvis Notre-Dame, 75004 Paris, France). *Ann. Gastroenterol. Hepatol.* 13(3):195-207; 1977.

0196 ROLE OF THE LIVER IN HORMONE METABOLISM AND IN REGULATION OF HORMONE CONTENT IN THE BLOOD. (Rus.) Ghenes, S. G. (Inst. Endocrinology and Hormone Chemistry, Kharkov, USSR). *Arkh. Patol.* 39(6):74-80; 1977.

0197 ULTRASTRUCTURAL CHANGES IN THE LIVER IN EXPERIMENTAL DIFFUSE PERITONITIS. (Rus.) Fishbein, A. V.; Olefir, V. P. (Medical Inst., Lvov, USSR). *Arkh. Patol.* 39(6):49-55; 1977.

0198 SUBMICROSCOPIC CHANGES OF GUINEA PIG HEPATOCYTES TREATED WITH HETEROLOGOUS MITOCHONDRIAL ANTIBODY. (Rus.) Barkhina, T. G.; Tomilets, V. A. (Res. Inst. Human Morphology, USSR Acad. Medical Sciences, Moscow, USSR). *Arkh. Patol.* 39(6):25-29; 1977.

0199 AUTOMATION ELEMENTS IN CONNECTION WITH AN EXPERIMENTAL ISOLATED LIVER. (Rus.) Shikhman, S. M.; Makshaev, I. I.; Potekhin, A. V. (Altai Medical Inst., USSR). *Eksp. Khir. Anesteziol.* (6):44-46; 1976.

0200 USE OF CHOLEDOCHOJEJUNAL FISTULA TO DETERMINE THE EXOCRINE FUNCTION OF THE LIVER. (Rus.) Tikhomirov, A. S.; Makarenko, Yu. I. (Vladivostok Medical Inst., Vladivostok, USSR). *Eksp. Khir. Anesteziol.* (6):43-44; 1976.

0201 EFFECT OF ENTERAL OXYGEN THERAPY AND VITAMIN E ON THE SECRETION AND CHEMICAL COMPOSITION OF BILE IN EXPERIMENTAL DYSTROPHY. (Rus.) Skakun, N. P.; Nesterovich, Ya. M. (Ternopol Medical Inst., Ternopol, USSR). *Vopr. Med. Khim.* 23(4):558-562; 1977.

0202 INVESTIGATION INTO THE ACTION OF ROENTGEN-CONTRAST MEDIA ON THE RESPIRATORY CHAIN OF MITOCHONDRIA IN THE LIVER OF RATS. (Rus.) Sergeev, P. V.; Obraztsov, N. V.; Timofeev, A. V.; Khalilov, E. M. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Farmakol. Toksikol.* 40(5): 591-595; 1977.

0203 TEMPORARY PORTACAVAL SHUNTING IN TWO-STAGE LIVER RESECTION. (Rus.) Rusanov, V. P.; Pogorelov, Yu. V.; Golovneva, V. I. (Semipalatinsk Medical Inst., Semipalatinsk, USSR). *Khirurgiia (Mosk.)* (7):20-23; 1977.

0204 EXPERIMENTAL INTENSIFICATION OF THE FUNCTIONAL ACTIVITY OF THE DONOR LIVER. (Rus.) Koperchak, A. M.; Babosh, Yu. L.; Peters, Yu. N. (Kemerovo Medical Inst., Kemerovo, USSR). *Khirurgiia (Mosk.)* (7):31-35; 1977.

0205 EXPERIMENTAL RHEOLOGY OF LIVER PARENCHYMA. (Ger.) Arnold, G.; Gressner, A. M. (Abteilung Anatomie der Rheinisch-Westfalischen Technischen Hochschule, Melatener Strasse 211, D-5100 Aachen, W. Germany). *Biomed. Tech.* 22(7/8): 182-186; 1977.

0206 α-FETOPROTEIN AND ALBUMIN SYNTHESIS BY HUMAN EMBRYO HEPATOCYTES. (Rus.) Eriser, T. L.; Elgort, D. A.; Abelev, N. I. (N. F. Gamaleya Inst. Epidemiology and Microbiology, USSR Acad. Medical Sciences, Moscow, USSR). *Biull. Eksp. Biol. Med.* 83(6):711-713; 1977.

0207 POSTVAGOTOMY DIVISION OF STELLATE RETICULOCYTES IN THE LIVER OF RATS. (Rus.) Astakhova, A. M.; Anufriev, B. T. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Biull. Eksp. Biol. Med.* 83(6):738-741; 1977.

0208 THE INFLUENCE OF SUBEXTREME AND EXTREME FACTORS OF THE HEPATIC LYSOSOMAL SYSTEM. (Rus.) Panin, L. E.; Mayanskaya, N. N. (Inst. Clinical and Experimental Medicine, Siberian Branch USSR Acad. Sciences, Novosibirsk, USSR). *Biull. Eksp. Biol. Med.* 83(6):678-680; 1977.

0209 REPLICATION OF DNA ASSOCIATED WITH NUCLEAR ENVELOPE OF REGENERATING RAT LIVER. (Rus.) Dvorkin, V. M.; Kiryanov, G. I.; Vanyshin, B. F. (A. N. Belozersky Lab. Molecular Biology and Bioorganic Chemistry, Moscow State Univ., Moscow, USSR). *Biokhimiia* 42(6):1001-1005; 1977.

0210 DISTURBANCE OF GLYCOLYTIC OXIREDUCTION IN THE LIVER DURING EXPERIMENTAL BURN DISEASE. (Rus.) Slobodin, V. B.; Lifshits, R. I.; Sobolevskaya, T. M. (Ivanovo Medical Inst., Ivanovo, USSR). *Biull. Eksp. Biol. Med.* 34(8):171-173; 1977.

0211 PECULIARITIES OF REGENERATION IN THE LIVER DEPRIVED OF POLYPLOID HEPATOCYTES. (Rus.) Dubovaya, T. K.; Kusch, A. A.; Brodsky, V. Ya. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Biull. Eksp. Biol. Med.* 34(8):221-224; 1977.

0212 DYNAMICS OF THE CHANGES IN BLOOD 11-OXYCORTICOSTEROIDS IN THE SUPRA- AND SUBHEPATIC FORMS OF PORTAL HYPERTENSION. (Rus.) Krivchik, A. A.; Mikhneva, L. M.; Abasheva, G. K. (Minsk Medical Inst., Minsk, USSR). *Patol. Fiziol. Eksp. Ter.* (4):79-82; 1977.

0213 LIVER OF NEWBORN RABBIT AFTER TRANSPLACENTAL EFFECT OF PHENOBARBITAL ADMINISTERED TO PREGNANT RABBIT DURING THE LAST PART OF PREGNANCY. (Pol.) Maryniak, R.; Dydyk, L. (Centrum Medycyny Doswiadczalnej i Klinicznej PAN, ul. Dworkowa 3, 00-784 Warszawa, Poland). *Patol. Pol.* 28(1):125-130; 1977.

0214 CHANGES IN THE ACTIVITY OF GLUTAMICO-ASPARTIC AND GLUTAMICO-ALANINE TRANSAMINASES IN THE LIVER OF RATS IN EXPERIMENTAL THERMAL TRAUMA. (Rus.) Kamilov, F. Kh.; Yakushev, V. S. (Chelyabinsk Medical Inst., Chelyabinsk, USSR). *Patol. Fiziol. Eksp. Ter.* (4):58-61; 1977.

See also, 0042, 0113, 0117, 0124, 0230, 0238, 0240, 0241, 0259, 0261, 0271, 0272, 0277, 0438, 0442, 0490, 0552, 0563, 0573, 0580, 0585, 0587, 0593, 0629, 0646, 0648, 0669, 0670, 0677, 0678, 0687, 0691, 0733.

SECRETION AND METABOLISM

Intestine

0215 THE ORIGIN OF CHYLOMICRON PHOSPHATIDYLCHOLINE IN THE RAT. (Eng.) Mansbach, C. M., II. (Veterans Admin. Hosp., Durham, NC 27710). *J. Clin. Invest.* 60(2):411-420; 1977.

Two pathways of origin of chylomicron phosphatidylcholine (CM PC), *de novo* synthesis and lyso PC-derived PC, were investigated using lymph and bile fistulated Sprague-Dawley rats infused with a stabilized triolein emulsion. To quantitate the contribution of PC generated by lyso PC acyltransferase to CM PC, [^{14}C-glycerol]PC was infused into the duodenum. The percentage of CM PC increased as the concentration of PC was increased in the infusate. Over 4-6 hr, virtually all the CM PC was derived from the PC infused when the infusate PC concentration was 10 mM. None of the infused [^{14}C-glycerol]PC was found in the aqueous phase of the gut luminal contents; i.e., there is minimal liberation of free PC glycerol within the intestinal lumen. There was less incorporation of radiolabel in the lymph subnatant PC, suggesting that CM and lymph subnatant PC are derived from different PC precursor pools. To determine the amount of CM PC synthesized from *de novo* sources, similarly prepared rats were infused with $^{32}P_i$. $^{32}P_i$ incorporation was used to represent *de novo* PC synthesis ultimately via the choline-dependent pathway. As the infusate PC concentration was increased, progressively less CM PC was derived from the infused $^{32}P_i$. Thus, exogenously infused PC inhibits *de novo* PC synthesis. When [^{32}P]bile PC and [^{14}C]-potato PC were infused into rats, the ratio of bile to potato derived PC in the CM averaged 1.72; i.e., bile PC was preferred as a CM PC precursor despite the greater similarity of the saturated fatty acids of potato PC to those of CM PC. The predominant saturated fatty acid of bile PC is palmitite, but palmitate and stearate are nearly equally present in CM PC, suggesting that the former cannot be the preferred precursor of the latter. The results suggest that PC synthesized from either pathway is preferentially distributed into the CM PC precursor pool, compared to the subnatant pool, and that these two pools are different.

0216 STEROL SYNTHESIS IN INTESTINAL VILLI AND CRYPT CELLS OF RATS AND GUINEA PIGS. (Eng.) Muroya, H.; Sodhi, H. S.; Gould, R. G. (Kyoto Univ. Faculty Medicine, Sakyo-ku, Kyoto, Japan). *J. Lipid Res.* 18(3):301-308; 1977.

The role of guinea pig and rat small intestinal villi and/or crypt cells in sterol synthesis was investigated. Three methods were used to measure sterol synthesis: incubation with ^{14}C-acetate of villi, crypt cell, and muscle fractions obtained by the method of graduated scraping, incubation of whole intestine pieces followed by fractionation into the same fractions, and *in vivo* studies in rats injected with ^{14}C-acetate and killed 7-40 min later, after which the intestine was fractionated by scraping or by serial sectioning of frozen tissue. All methods were in agreement in showing sterol synthesis in villi to be as active per mg protein as in crypt cells. Measurements of the isotope content of the total nonsaponifiable lipid fractions led to the same conclusions as did the isotope content of the digitonin-precipitable sterol fractions. For a given period of incubation, the ratio of the two values was the same in villi and crypts, ruling out the possibility of synthesis of sterols in crypts prior to transfer to villi. The fractionation into villi, crypts, and muscle fractions by scraping greatly reduced the total sterol synthetic activity in rat but not in guinea pig small intestine, indicating the hazard of using only this *in vitro* technique to make quantitative estimates of the sterol synthetic capacity of tissue fractions.

0217 EFFECT OF SMALL BOWEL RESECTION ON GASTRIN RELEASE, CATABOLISM, AND ACTIVITY IN DOGS. (Eng.) Woussen-Colle, M. C.; Brihaye, Cl.; Limbosch, J. M.; De Graef, J. (Hopital Universitaire Saint-Pierre, Rue Haute, 322, B. 1000 Brussels, Belgium). *J. Surg. Res.* 21(4):225-232; 1976.

The effects of upper small bowel resection on the catabolism and activity of gastrin were determined in three dogs prepared with a vagally denervated Heidenhain pouch (HP) and a gastric fistula (GF). Conditions prior to testing varied: starvation, feeding, and infusion of exogenous gastrin. All tests were made before and 1 and 6 months post-resection (PR) of the upper half of the small bowel starting 10 cm from the ligament of Treitz. Continuity was restored by a two-layer end-to-end bowel anastomosis. At 1 month PR, peak acid output over 30 min from the HP was (in mEq, pre-resection values in parentheses): 2.17 ± 0.26 (1.73 ± 0.30), $p<0.05$, in response to exogenous gastrin and 3.62 ± 0.25 (2.20 ± 0.44), $p<0.01$, in response to feeding. The total acid output over 5.5 hr was 29.38 ± 1.67 (12.72 ± 2.41), $p<0.01$. At 6 months PR, the respective values were 1.89 ± 0.26 ($p>0.05$), 2.62 ± 0.35 ($p>0.05$), and 22.23 ± 3.12 ($p<0.01$). At 1 month PR, immunoreactive serum gastrin levels were 131.7 ± 28.3 pg/ml (41.8 ± 4.0), $p<0.01$; peak gastrin concentration after feeding was 478.3 ± 47.0 pg/ml (144.1 ± 25.0), $p<0.01$, and the integrated gastrin response after feeding was 59.3 ng/ml/5 hr (11.4 ± 2.2), $p<0.01$. At 6 months PR, these values were 65.9 ± 3.7 pg/ml ($p<0.01$), 227.3 ± 33.2 ($p<0.05$), and 24.7 ± 4.0 ng/ml/5 hr ($p<0.01$). The metabolic clearance rate of exogenous gastrin before resection and 1 and 6 months PR was 95.2 ± 11.9, 68.6 ± 8.8 ($p<0.05$), and 107.5 ± 11.2 ($p>0.05$) ml/kg/min, resp. The increased acid response from the HP to a meal PR is due to increased reactivity and increased stimulation of the parietal cells. These effects could be related to the removal of intestinal inhibitors acting on parietal and G cells and/or to the interruption of inhibitory neural reflexes between the small bowel and stomach. In dogs, the small bowel appears to control gastric secretion and gastrin release by humoral and/or neural mechanisms. PR, the mechanism is impaired, but a progressive adaptation takes place, partly as a result of hyperplasia (in the remaining small bowel) of cells catabolizing gastrin and those secreting enterogastrones.

0218 THE INTESTINAL BRUSH BORDER MEMBRANE IN DIABETES: STUDIES OF SUCRASE-ISOMALTASE METABOLISM IN RATS WITH STREPTOZOTOCIN DIABETES. (Eng.) Olsen, W. A.; Korsmo, H. (William S. Middleton Memorial Veterans Admin. Hosp., Madison, WI 53706). *J. Clin. Invest.* 60(1):181-188; 1977.

To determine the mechanism by which diabetes stimulates the functional activity of the intestinal brush border membrane, the effects of streptozotocin diabetes on the metabolism of one membrane protein, sucrase-isomaltase, was studied in male Sprague-Dawley rats. The protein was purified, and an anti-serum was prepared. Sucrase-isomaltase from control and diabetic rats was immunologically identical as shown by Ouchterlony double-diffusion analysis of papain-solubilized mucosal proteins. The increase in sucrase enzyme activity in diabetic animals (31.0 ± 1.4 U 5 days after streptozotocin versus 13.1 ± 1.0 in controls) was the consequence of increased enzyme protein and was not an alteration in catalytic efficiency, as demonstrated by quantitative immunoprecipitin reactions. To account for increased sucrase-isomaltase protein in diabetes, papain-solubilized mucosal proteins labeled by injection of ^{14}C-carbonate and ^{14}C-leucine were studied. Diabetes did not affect the decay of labeled total protein, but prolonged the decay of labeled sucrase-isomaltase. The half time of sucrase-isomaltase was 4.4 hr in control animals after ^{14}C-carbonate injection and 8.8 and 10.2 hr 2 and 5 days, resp., after induction of streptozotocin diabetes. Similar results were obtained in experiments with ^{14}C-leucine with diabetes increasing the half-time from 6 to 13.6 hr. Diabetes did not appear to increase the rate of addition of sucrase-isomaltase to the brush border membrane, since it did not affect the 10- and 60-min incorporations of isotope into sucrase-isomaltase protein relative to incorporation into total protein and did not alter rate constants for synthesis calculated from the half-time and the change in enzyme mass over time. Thus, enhanced sucrase activity in the diabetic animal is the consequence of an increase in sucrase-isomaltase protein, which develops because of a decrease in its rate of degradation.

0219 SOME OBSERVATIONS ON THE DETERMINATION OF EXTRACELLULAR FLUID VOLUME OF JEJUNAL TISSUE USING [^{3}H]INULIN AND [^{14}C]INULIN. (Eng.) Dinda, P. K.; Beck, I. T.; Beck, M. (Hotel Dieu Hosp., Kingston, Ontario, Canada). *Can. J. Physiol. Pharmacol.* 55(3):389-393; 1977.

The nature of equilibration of [^{3}H]inulin and [^{14}C]-inulin was examined in the *in vitro* hamster jejunum. Rings of everted jejunum were incubated at 37 C in Krebs-Ringer bicarbonate solution containing 10 mM glucose, tracer amounts of [^{14}C]inulin, and tracer amounts of [^{3}H]inulin from one of the two lots (lot X and lot Y) tested. The incubations were carried out for 5, 10, 20, 30, 45, or 60 min. One lot of [^{3}H]inulin (lot X) provided an estimate of the extracellular (EC) fluid volume which, at all periods of incubation, was comparable with that provided by the [^{14}C]inulin. In contrast, the estimate of EC fluid volume obtained from the other lot of [^{3}H]-inulin (lot Y) was consistently higher than that obtained from the [^{14}C]inulin and increased linearly with the period of incubation. Because, with [^{3}H]-inulin of lot Y, the calculated intracellular (IC) fluid volume decreased linearly with the period of incubation, and because this was not the case with the [^{14}C]inulin or with the [^{3}H]inulin of lot X, it appears that [^{3}H]inulin of lot Y failed to equilibrate within the tissue water, while the other lot of [^{3}H]inulin (lot X), as well as [^{14}C]-inulin, did. Although the EC fluid volume obtained from [^{14}C]inulin (as well as that from [^{3}H]-inulin of lot X) increased progressively, the rate

of increase during the first 30 min was considerably higher than that during the last 30 min of incubation. The progressive increase in the $[^{14}C]$-inulin space, however, was accompanied by a corresponding increase in the total water content as well as in the calculated IC fluid content of the tissue. Since the IC fluid volume as percentage of total water content after 5 min of incubation was not significantly different from that after 60 min of incubation, it appears that $[^{14}C]$inulin equilibrates within the EC fluid after 5 min, and that the progressive increase in the calculated EC fluid volume is the result of a corresponding increase in the EC fluid (containing $[^{14}C]$inulin) content of the tissue.

0220 DIFFERENTIATION OF EPITHELIAL CELLS IN HUMAN JEJUNUM: LOCALIZATION AND QUANTIFICATION OF AMINOPEPTIDASE, ALKALINE PHOSPHATASE AND ALDOLASE ISOZYMES IN TISSUE SECTIONS. (Eng.) Wachsmuth, E. D. (Friedrich Miescher-Institut, P.O. Box 273, CH-4002 Basel, Switzerland). *Histochemistry* 48(2):101-109; 1976.

Aminopeptidase, alkaline phosphatase, and aldolase isozyme activities were studied in human jejunal crypt cells at different stages of differentiation. Jejunal sections were stained histochemically for aminopeptidase and alkaline phosphatase, and the aldolase isozymes were detected with the mixed aggregation immunocytochemical technique. All enzyme concentrations increased from the bottom to the upper part of the crypt. The concentration of aldolase-A per cell was the same in the upper part of the crypt and the villus, whereas the concentration of the other three enzymes was still higher. Therefore, high amounts of aldolase-B, aminopeptidase, and alkaline phosphatase are present in cells highly active in absorption in a fashion similar to that found in the proximal tubule cells of kidney. The relatively undifferentiated cells of the crypts contained both aldolase-A and aldolase-B. Alkaline phosphatase gains its full activity later than aminopeptidase. The synthesis of microvillar membrane enzymes comes to an end earlier than that of the cytosol enzymes.

0221 HCO_3^- AN H^+ SECRETION IN RAT ILEUM IN VIVO. (Eng.) Podesta, R. B.; Mettrick, D. F. (Dept. Zoology, Univ. Toronto, Ontario M5S 1A1, Canada). *Am. J. Physiol.* 232(6):E574-E579; 1977.

Ion transport was studied in the *in vivo* rat ileum. Replacing Cl^- in the perfusion fluids decreased the luminal pH of perfused ileum. Varying the initial pH in Cl^--depletion studies indicated an equilibrium pH near 6.6. Na^+ depletion in Cl^--depleted fluids partially inhibited luminal acidification. Addition of HCO_3^- accelerated Na^+ absorption and decreased Cl^- absorption. Luminal PCO_2 was increased using fluids buffered to initial pH 6.6, suggesting secretion of HCO_3^- rather than OH^-. The decreased HCO_3^- secretion using Cl^--depleted fluids was not due to an accelerated rate of H^+ secretion. Reaffirmed and expanded in this study is an ileal H^+ secretory process that is partially related to Na^+ absorption and direct HCO_3^- secretion that is related to Cl^- and Na^+ absorption.

0222 Ca IONOPHORE-STIMULATED ION SECRETION IN RABBIT ILEAL MUCOSA: RELATION TO ACTIONS OF CYCLIC 3',5'-AMP AND CARBAMYLCHOLINE. (Eng.) Bolton, J. E.; Field, M. (Beth Israel Hosp., Boston, MA 02215). *J. Membr. Biol.* 35(2):159-173; 1977.

The possibility that calcium acts as a mediator of secretion in the small intestine was investigated, and experiments were carried out to determine whether cholinergic agonists or serotonin act as calcium ionophores. Addition of the Ca ionophore A23187 (0.5 μg/ml) to the serosal side of stripped rabbit ileal mucosa produced changes in ion transport qualitatively identical with those produced by cyclic 3',5'-AMP (cAMP) and theophylline: an increase in short-circuit current and resistance, net secretion of Cl due both to a decrease in the unidirectional mucosa (m) to serosa (s) flux and to an increase in the s to m flux, and net secretion of Na due to a decrease in m to s flux. Measurements of intracellular cAMP level demonstrated no change following incubation with the ionophore. Removal of Ca from the serosal bathing medium diminished the effects of A23187 but did not impair the action of theophylline. Removal of Ca from both the mucosal and serosal bathing media by replacing it with Sr completely abolished the potential difference response to A23187. These results suggest that the ionophore elicits its secretory actions by decreasing Ca influx into the epithelial cells. In a similar way, carbamylcholine and serotonin, secretagogues known to have no effect on intracellular cAMP level in intestinal mucosa, were shown to be dependent on extracellular Ca to produce their full electrical response. In contrast, the secretagogues vasoactive intestinal peptide and prostaglandin E_1, which raise cAMP concentration in intestinal mucosa, do not appear to require external Ca. Ca may be an intracellular mediator of intestinal ion and water secretion, and some intestinal secretagogues may act as Ca ionophores.

0223 ISOLATION AND IMMUNOCHEMICAL AND CHEMICAL CHARACTERIZATION OF CARCINOEMBRYONIC ANTIGEN-LIKE SUBSTANCES IN COLON LAVAGES OF HEALTHY INDIVIDUALS. (Eng.) Egan, M. L.; Pritchard, D. G.; Todd, C. W.; Go, V. L. W. (Dept. Microbiology, Univ. Alabama in Birmingham, University Station, Birmingham, AL 35294). *Cancer Res.* 37(8):2638-2643; 1977.

Material with carcinoembryonic antigen (CEA)-like activity, isolated from colon lavages of healthy individuals, was chemically characterized. The CEA-like activity was purified by gel filtration on Sepharose 6B and Sephadex G-200 and by affinity chromatography on concanavalin A linked to Sepharose. The purified material migrated in polyacrylamide-sodium dodecyl sulfate electrophoresis as a single diffuse band with mobility identical with that of

tumor CEA. The material with CEA activity from tumor tissue and from normal colon lavages gave lines of identity in double diffusion experiments and had similar inhibition curves with essentially the same specific activities in a radioimmune assay for tumor CEA. The amino acid and carbohydrate compositions were similar to that observed for CEA tumor preparations. Lysine was the single amino terminus. Methylation analysis demonstrated that the monosaccharide linkages were similar to those in CEA isolated from tumor tissue. The data support the view that CEA is not restricted to fetal and neoplastic tissue but is also present in healthy adult tissue.

0224 FUNCTIONAL AND MORPHOLOGICAL STUDIES ON INTESTINAL MUCOSA OF THE RAT UNDER CHRONIC GLUCAGON APPLICATION. (Ger.) Lorenz-Meyer, H.; Menge, H.; Riecken, E. O. (Medizinische Universitatsklinik, Mannkopffstrasse 1, D-3550 Marburg/Lahn, W. Germany). *Res. Exp. Med.* 170(2):181-192; 1977.

0225 RECEPTORS FOR VASOACTIVE INTESTINAL PEPTIDE (VIP) IN ENTEROCYTES: SPECIFIC BINDING AND STIMULATION OF CYCLIC AMP. (Fre.) Laburthe, M.; Besson, J.; Hui Bon Hoa, D.; Rosselin, G. (Hopital Saint-Antoine, 184, rue du Faubourg Saint-Antoine, 75571 Paris Cedex 12, France). *C. R. Acad. Sci.* [*D*] *Paris* 284(21):2139-2142; 1977.

0226 INFLUENCE OF DIETARY LACTOSE ON THE INITIATION OF LACTOSE ACTIVITY IN THE DIGESTIVE TRACT OF PIGS. (Fre.) Aumaitre, A.; Fevrier, C.; Dewulf, H. (Station de Recherches sur l'Elevage des Porcs, 78350 Jouy-en-Josas, France). *C. R. Acad. Sci.* [*D*] *Paris* 284(21):2135-2138; 1977.

0227 EFFECT OF DIETS OF VARYING PROTEIN CONTENT ON NUCLEIC ACIDS OF THE INTESTINAL MUCOSA. (Ukr.) Malyzheva, T. D.; Okunev, V. N. (Inst. Nutrition Hygiene, Kiev, USSR). *Fiziol. Zh.* 22(4):518-522; 1976.

0228 COMPARATIVE ANALYSIS OF CELL POPULATION OF ENTEROCYTES IN VARIOUS PARTS OF THE SMALL AND LARGE INTESTINE OF RATS. I. PARAMETERS OF THE MITOTIC CYCLE AND HETEROGENEITY OF THE PROLIFERATING ENTEROCYTE POPULATION. (Rus.) Pozharissky, K. M.; Klimashevsky, V. F.; Gushchin, V. A. (Res. Inst. Oncology, Leningrad, USSR). *Tsitologiia* 19(3):303-317; 1977.

0229 COMPARATIVE ANALYSIS OF CELL POPULATION OF ENTEROCYTES IN VARIOUS PARTS OF THE SMALL AND LARGE INTESTINE OF RATS. II. THE RATE OF ENTEROCYTE MIGRATION: GENERAL CONCLUSION AND DISCUSSION. (Rus.) Pozharissky, K. M.; Klimashevsky, V. F.; Gushchin, V. A. (Res. Inst. Oncology, Leningrad, USSR). *Tsitologiia* 19(3):318-328; 1977.

0230 EVIDENCE OF AN ENTEROHEPATIC CYCLE FOR S-CARBOXYMETHYLCYSTEINE. (Fre.) Servin, A. (Laboratoire Joullie, 20 bis, quartier Boieldieu, La Defense, 92803 Puteaux, France). *C. R. Soc. Biol. (Paris)* 171(1):39-42; 1977.

See also, 0002, 0008, 0011, 0014, 0017, 0019, 0021, 0039, 0046, 0113, 0119, 0232, 0233, 0235, 0236, 0237, 0245, 0250, 0263, 0270, 0271, 0280, 0281, 0350, 0428, 0435, 0765.

GENERAL

0231 ULTRASTRUCTURAL CHANGES IN THE GASTRIC MUCOSA FOLLOWING HEMORRHAGIC SHOCK IN PIGS. (Eng.) Collan, Y.; Kivilaakso, E.; Kalima, T. V.; Lempinen, M. (Second Dept. Pathology, Univ. Helsinki, Helsinki, Finland). *Circ. Shock* 4(1):13-25; 1977.

Ultrastructural changes in the gastric mucosa of 14 piglets subjected to transient hemorrhagic shock (3-hr duration, mean arterial pressure 40 mm Hg) were studied. After 30 min, there was mucosal edema and extravasation of RBC. Microthrombi with degranulating thrombocytes and fibrin strands were seen in the capillaries. These changes were also seen in the antral mucosa. At this stage the cells of the neck region of the gastric glands showed mitochondrial dilatation, and their apical parts were seen to bulge into the lumen of the gastric glands. Other cell types were affected later and necrosis progressed from the surface into deeper parts of the mucosa. Mast cells and mucosal endocrine cells were relatively resistant, being mostly undamaged at the end of the shock period and later. The findings suggest that, during the shock, local formation of thrombi contributes to mucosal ischemia and ulcer formation, and that the cells at the neck region of the gastric glands form the locus of limited resistance to ulcerogenic effects.

0232 'LUMINAL NUTRITION' VERSUS 'FUNCTIONAL WORK-LOAD' AS CONTROLLERS OF MUCOSAL MORPHOLOGY AND EPITHELIAL REPLACEMENT IN THE RAT SMALL INTESTINE. (Eng.) Clarke, R. M. (Faculty

Medicine, Univ. Newcastle, Newcastle, N.S.W. 2308, Australia). *Digestion* 15(5):411-424; 1977.

The relation of small intestinal morphology and epithelial replacement to the secretory and absorptive workload that the epithelium performs was investigated in albino male Wistar rats. Isotonic solutions of substrate were infused into surgically unrestrained rats that fed normally via the "intestine-in-continuity." Infusion of glucose, galactose, or NaCl all caused a local increase in villus height, while α-methyl glucoside and mannose had no effect. Glucose, galactose, and α-methyl glucoside all stimulated a similar increase in counts of vincristine metaphases in microdissected crypts, although the absolute level of cell production was lower throughout the gut in glucose-infused rats. Sodium chloride infusion also caused a small stimulation of cell production locally, but a considerable increase in the uninfused intestine-in-continuity. Mannose infusion had a variable effect on cell nutrition" (nutrients in the lumen) as a controller of epithelial replacement in the rat small intestine, and suggest that a mechanism based on the workload of the epithelium may be more appropriate.

0233 THE PRODUCTION OF AN UNUSUAL TISSUE REACTION IN RABBIT BOWEL INJECTED WITH CROHN'S DISEASE HOMOGENATES. (Eng.) Simonowitz, D.; Block, G. E.; Riddell, R. H.; Kraft, S. C.; Kirsner, J. B. (Dept. Surgery, Univ. Chicago, 950 E. 59th St., Chicago, IL 60637). *Surgery* 82(2):211-218; 1977.

Attempts were made to reproduce evidence of a transmissible factor in Crohn's disease. Twenty New Zealand White rabbits received inoculum of tissue homogenates from the terminal ileum of normal subjects and patients with ulcerative colitis or Crohn's disease. This inoculum was injected into the wall of the ascending colon, and the animals were examined after 1 yr. Changes were noted consistently only in those animals receiving the Crohn's disease inoculum. These changes consisted of thickened bowel wall with increased mucosal folds, thickened mesenteric fat with some creeping, irregular areas of thin colonic mucosa, and hyperplastic mesenteric lymph nodes. The terminal ileum distant from the inoculum site was smooth with atrophic changes only in the Crohn's inoculated group of animals. These animals also had discrete collections of macrophages in colon or sacculus, mucosal and submucosal edema, and chronic inflammation of the colon. However, the characteristic features of Crohn's disease were not reproduced.

0234 MALIGNANT DEGENERATION OF EXPERIMENTAL ULCERATIVE COLITIS OF THE RAT FOLLOWING SUBCUTANEOUS INJECTION OF 1,2-DIMETHYLHYDRAZINE. (Ger.) Dahm, K.; Knipper, A.; Mitschke, H. (Abteilung Allgemeinchirurgie der Chirurgischen Klinik, Martinistr. 52, D-2000 Hamburg 20, W. Germany). *Langenbecks Arch. Chir.* 343(4):307-311; 1977.

Experiments were carried out in male Wistar rats to determine whether the colon is more susceptible to malignant transformation in the presence of ulcerative colitis than when it is intact. Ulcerative colitis was induced by treating the rats with 4% carrageenan in the drinking water. The colon-specific carcinogen 1,2-dimethylhydrazine (DMH, 19.3 mg/kg) was then administered s.c. daily for 7 weeks. The animals received carrageenan up until 6 weeks after the last injection of DMH. Seventeen of the 18 rats with carrageenan-induced ulcerative colitis developed multiple adenocarcinomas of the distal colon by 15 weeks after the last DMH injection. In addition to adenocarcinomas, squamous cell metaplasias were also present. Only 3 of 18 controls treated with DMH alone developed adenocarcinomas of the distal colon, and none of 12 animals treated with carrageenan alone developed adenocarcinomas; 1 of the 12 animals did develop an adenoma with marked dysplasia. The results suggest that, during ulcerative colitis, the rat colon is more susceptible to tumor induction than it is when intact.

0235 DIFFERENCES IN SURVIVAL OF MOUSE COLON CRYPTS AFTER WHOLE- OR PARTIAL-BODY IRRADIATION. (Eng.) Hamilton, E. (Imperial Cancer Res. Fund Lab., Lincoln's Inn Fields, London WC2A 3PX, England). *Int. J. Radiat. Biol.* 31(4):341-347; 1977.

The survival of mouse colon crypts after whole- and partial-body x-irradiation was studied by the microcolony technique. The D_0 for crypt survival was 266 rad after whole-body irradiation in air, but when the colon alone was irradiated, the D_0 was 340 rad. In mice that were breathing 95% oxygen, the D_0 values were 238 rad for whole-body and 302 rad for colon irradiation. The survival curves were all extrapolated to the same number, and the threshold dose was also increased with colon irradiation. There was therefore an enhancement of colon crypt survival of about 27% by local as opposed to whole-body irradiation. These results might be explained by a circulating repair promoter or by the production of a toxin after whole-body irradiation.

0236 ISOLATION OF THE CECAL MICROFLORA OF MICE AND COMPARISON BETWEEN THE GASTRO-INTESTINAL AND THE FECAL MICROFLORAS. (Eng.) Koopman, J. P.; Janssen, F. G. J.; van Druten, J. A. M. (Central Animal Lab., Catholic Univ. Nijmegen, Geert Grooteplein Noord 29, Nijmegen, Netherlands). *Z. Versuchstierkd.* 19(1/2):62-70; 1977.

The number of bacteria that could be cultured from the cecum of SPF mice was determined, and the gastrointestinal and fecal microfloras were compared. The recovery of cecum bacteria was determined by three bacteriological techniques: an aerobic technique, an anaerobic jar technique, and a strict anaerobic glove-box technique. The median values for the yields were 0.46%, 4.8%, and 37%, resp. Lactobacilli, staphylococci, and streptococci were present in relatively low levels (median values/g, resp., 9.3×10^8, 1.6×10^6, and 2.4×10^5). Subsequently,

the recovery of cecum bacteria from CRF-mice and GF-mice associated with 10 *in vitro* cultured anaerobic (bacterial) strains (ex-GF) was determined using a strict anaerobic technique. These yields were compared with the results obtained in SPF-mice. The cecal weights were different. However, the microscopic counts per cecum were not significantly different among the three groups, i.e., 1.6×10^{10} (median values). Accordingly, the concentration of cecal bacteria was not equal in the three groups. The viable count as percentage of the total numbers of bacteria in the ceca of ex-GF, CRF, and SPF-mice was 54%, 44%, and 37%, resp. Within the three bacteriologically different groups, evidence for a positive correlation between cecal weight and microscopic count per cecum, resp., viable count per cecum was found. The bacterial concentrations per gram contents in stomach/small intestine, cecum/large intestine and feces were 2.1×10^{8}, 1.7×10^{10}, and 1.9×10^{10}, resp. The excretion of bacteria with the feces per 24 hr varied between 54% and 400% (median value 150%) of the number of bacteria present in the gastrointestinal tract.

0237 THE INFLUENCE OF ADRENOCEPTOR ACTIVITY ON CELL PROLIFERATION IN COLONIC CRYPT EPITHELIUM AND IN COLONIC ADENOCARCINOMATA. (Eng.) Tutton, P. J. M.; Barkla, D. H. (Dept. Anatomy, Monash Univ., Clayton, Victoria, 3168, Australia). *Virchows Arch.* [*Cell Pathol.*] 24(2):139-146; 1977.

The effects of chemical sympathectomy and of the injection of amines or amine receptor blocking drugs on cell proliferation in colonic crypts and in dimethylhydrazine-induced colonic carcinomas were examined in male Sprague-Dawley rats using a stathmokinetic technique. In animals chemically sympathectomized by the injection of 6-hydroxydopamine (100 mg/kg, i.v.), cell proliferation essentially ceased in the colonic crypts but continued at a normal rate in the tumors. Stimulation of α-adrenoceptors by metaraminol (2.5 mg/kg) caused acceleration of cell proliferation in colonic crypts but not in tumors. Conversely, blockade of α-adrenoceptors by phentolamine (10 mg/kg) inhibited cell proliferation in crypts but not in tumors. The injection of epinephrine (1 mg/kg) inhibited cell proliferation in the tumors but not in colonic crypts, whereas blockade of β-adrenoceptors by propranolol (20 mg/kg) accelerated cell proliferation in tumors but not in colonic crypts. It is postulated that cell proliferation in the crypts of Lieberkuhn in rat colon resembles that in rat jejunum in being controlled by the autonomic nervous system. However, tumor cell proliferation does not appear to be subject to such regulation.

0238 PATHOGENESIS OF SHIGELLA DIARRHEA. VII. EVIDENCE FOR A CELL MEMBRANE TOXIN RECEPTOR INVOLVING β1→4-LINKED n-ACETYL-D-GLUCOSAMINE OLIGOMERS. (Eng.) Keusch, G. T.; Jacewicz, M. (Mount Sinai Sch. Medicine, City Univ. New York, New York, NY 10029). *J. Exp. Med.* 146(2):535-546; 1977.

To determine whether mammalian cells possess a membrane receptor for *Shigella dysenteriae* 1 cytotoxin, the binding of this cytotoxin to HeLa cells in culture and to isolated rat liver cell membranes was studied by an indirect consumption assay of toxicity from the medium or by determination of cytotoxicity to the HeLa cell monolayer. Both liver cell membranes and HeLa cells removed toxicity from the medium during incubation, in contrast to WI-38 and Y-1 mouse adrenal tumor cells. Uptake of toxin was directly related to concentration of membranes added, time, and temperature, and indirectly related to the ionic strength of the buffer used. The chemical nature of the membrane receptor was characterized by using three principal approaches: (a) enzymatic sensitivity, (b) competitive inhibition, and (c) receptor blockade studies. The receptor was destroyed by proteolytic enzymes, phospholipases (which markedly altered the gross appearance of the membrane preparation), and by lysozyme, but not by a variety of other enzymes (galactose oxidase, β-galactosidase, neuraminidase). Of 28 carbohydrate and glycoprotein haptens studied, including cholera toxin and ganglioside, only the chitin oligosaccharide lysozyme substrates, per *N*-acetylated chitotriose, chitotetraose, and chitopentaose, were effective competitive inhibitors. Greatest inhibition was found with the trimer, *N*, *N'*,*N"* triacetyl chitotriose. Of three lectins studied as possible receptor blockers, including phytohemagglutinin, concanavalin A, and wheat germ agglutinin, only the latter, which is known to possess specific binding affinity for *N*,*N'*,*N"* triacetyl chitotriose, blocked toxin uptake. Evidence from all three approaches indicate that a glycoprotein toxin receptor exists on mammalian cells, with involvement of oligomeric β1→4-linked *N*-acetyl glucosamine in the receptor. This receptor is clearly distinct from the G_{M1} ganglioside thought to be involved in the binding of cholera toxin to the cell membrane of a variety of cell types susceptible to its action.

0239 HEPATIC BLOOD FLOW MEASUREMENT: A COMPARISON OF THE INDOCYANINE GREEN AND ELECTROMAGNETIC TECHNIQUES IN NORMAL AND ABNORMAL FLOW STATES IN THE DOG. (Eng.) Teranaka, M.; Schenk, W. G. (Dept. Surgery, State Univ. New York at Buffalo, Buffalo, NY). *Ann. Surg.* 185(1):58-63; 1977.

Hepatic blood flow was measured simultaneously by the electromagnetic technique (EMF) and by clearance of a single injection of indocyanine green (ICG, 0.4-0.7 mg/kg body weight) in 9 normal dogs and 14 dogs in unsteady state following hemorrhage (the amount of acute blood loss ranged 0.5-1.0% of body weight) to establish the validity of the ICG method in both steady and unsteady states. Close correlation between the ICG clearance (25.3 ± 9.7 ml/kg body weight/min) technique and the EMF technique (26.3 ± 9.1 ml/kg body weight/min) under normal flow states was seen. However, under the unsteady condition (15 min bleeding), ICG indirect flow measurement (14.04 ± 4.8 ml/kg body weight/min) estimated only 71% of the actual instantaneous

electromagnetic flow value of 19.0 ± 4.9 ml/kg body weight/min. It is suggested that while the EMF method continues to measure the actual total hepatic blood flow, the ICG method may measure the functional hepatic flow.

0240 EFFECTS OF BILE AND BILE ACIDS ON CULTURED HUMAN FIBROBLASTS. (Eng.) Trias, X.; Strebel, H. M.; Paumgartner, G.; Wiesmann, U. N. (Dept. Pediatrics, Univ. Bern, Freiburgstrasse 23, CH-3010 Berne, Switzerland). *Eur. J. Clin. Invest.* 7(3):189-194; 1977.

To determine whether impaired healing induced by leakage of bile is a factor responsible for complications after reconstructive bile duct surgery, the cytotoxicity of human bile, and its major bile acids, on cultured human fibroblasts was studied. The effects of bile and bile acids on cell morphology and growth, on synthesis and secretion of $^{35}SO_4$-mucopolysaccharides, and on release of a lysosomal enzyme were evaluated. Normal human fibroblasts derived from a standard culture strain (MRC-5) were grown to confluence and exposed to: (a) sterile human T-tube bile, (b) a mixture of bile acids resembling that of human bile, or (c) various concentrations of the glycine and taurine conjugates of cholic, chenodeoxycholic, or deoxycholic acid. Medium containing whole bile (total bile acid concentration 0.25, 0.75, or 1.6 mM) exerted time- and dose-dependent cytotoxic effects on morphology and growth and release of lysosomal enzyme. Synthesis and secretion of $^{35}SO_4$-mucopolysaccharides were markedly inhibited. The bile acid mixture exhibited the same time- and dose-dependent effects. The conjugates of deoxycholic acid were found to be the most toxic of the individual bile acids studied.

0241 THE ROLE OF TUBULAR REABSORPTION IN THE RENAL EXCRETION OF BILE ACIDS. (Eng.) Barnes, S.; Gollan, J. L.; Billing, B. H. (Univ. Alabama Medical Center, Birmingham, AL 35294). *Biochem. J.* 166(1):65-73; 1977.

To provide possible explanations for the bile acid composition in human cholestatic urine, the renal excretion of bile acids was studied in an isolated rat kidney preparation perfused with a protein-free medium. Tubular reabsorption exceeded 95% for both nonsulfated and sulfated bile acids at filtered loads of less than 30 nmol/min. At low filtered loads, the reabsorption of taurocholate and taurochenodeoxycholate was almost complete. Efficient reabsorption of taurochenodeoxycholate was maintained over a wider range of filtered loads than for taurocholate. These observations suggest that active transport may occur. At high filtered loads, saturation of reabsorption of taurocholate and taurochenodeoxycholate did not occur, which indicates that passive diffusion is involved in reabsorption. Active proximal-tubular secretion of bile acids was not demonstrated in competition experiments with *p*-aminohippurate. The fractional reabsorption of taurocholate, chenodeoxycholate, 3,7-disulfate, and chenodeoxycholate 7-monosulfate was decreased by the addition of taurochenodeoxycholate to the perfusate, so that their renal excretion was enhanced. This interaction between the bile acids for reabsorption may explain the different composition of bile acids in urine compared with that in plasma in cholestasis in man. Conjugated bilirubin decreased the fractional reabsorption of both taurocholate and taurochenodeoxycholate at low filtered loads (<30 nmol/min) but not at high filtered loads (400 nmol/min).

0242 ROLE OF INTRACELLULAR SPACE IN THE INTEGRATION OF NERVOUS AND HORMONAL REGULATION OF THE DIGESTIVE TRACT. (Rus.) Solov'eva, I. A. (No affiliation given). *Dokl. Akad. Nauk SSSR* 233(6):1208-1210; 1977.

0243 DUODENAL FISTULA: LONG-TERM OBSERVATIONS IN ANIMAL EXPERIMENTS. (Ger.) Helwing, E. (Medizinische Hochschule Hannover, Pasteurallee, D-3000 Hanover 51, W. Germany). *Z. Exp. Chir.* 10(2):79-83; 1977.

0244 STRUCTURAL AND FUNCTIONAL CHANGES IN THE SMALL INTESTINE IN PERITONITIS. (Rus.) Popov, V. A. (S. M. Kirov Military Medical Acad., Leningrad, USSR). *Patol. Fiziol. Eksp. Ter.* (1): 32-37; 1976.

0245 THE INTERACTION OF THE INTESTINAL EPITHELIUM WITH *Vibrio cholerae* AND *Escherichia coli* (HISTOLOGICAL AND ELECTRON MICROSCOPIC STUDY). (Rus.) Polotsky, Yu. E.; Dragunskaya, E. M.; Snigirevskaya, E. S.; Seliverstova, V. G. (Inst. Experimental Medicine, USSR Acad. Medical Sciences, Leningrad, USSR). *Arkh. Patol.* 39(2):10-17; 1977.

0246 MORPHOLOGICAL MANIFESTATIONS OF ABSORPTION OF *Shigella sonnei* ON THE INTESTINAL EPITHELIUM. (Rus.) Kovalchuk, V. K. (Kiev Scientific Res. Inst. Infectious Diseases, Kiev, USSR). *Arkh. Patol.* 39(4):68-73; 1977.

0247 ANASTOMOSIS DELAYING THE ALIMENTARY TRANSIT BY 180° AXIAL ROTATION OF THE UPPER INTESTINAL SEGMENT FOLLOWING MASSIVE RESECTION OF THE SMALL INTESTINE. PRELIMINARY REPORT ON EXPERIMENTS ON ADULT DOGS. (Fre.) Salomao, J.; Bosgiraud, F.; Vayre, P. (Hopital Vaugirard 389, rue de Vaugirard, 75 Paris, France). *J. Chir. (Paris)* 112(1/2):99-105; 1976.

0248 EXPERIMENTAL STUDY OF EVERTED INTESTINAL CLOSURES. COMMUNICATION I: ILEOILEAL ANASTOMOSIS. (Ita.) Dell'Anna, B.; Sacco, M.; Melissari, M. (Istituto di Clinica Chirurgica Generale e Terapia Chirurgica II[a], Universita degli Studi Di Parma, Parma, Italy). *Chir. Patol. Sper.* 24(4):289-306; 1976.

0249 EXPERIMENTAL STUDY OF EVERTED INTESTINAL CLOSURES. COMMUNICATION II: COLONIC COLOCOLIC ANASTOMOSIS. (Ita.) Dell'Anna, B.; Sacco, M.; Melissari, M. (Istituto di Clinica Generale e Terapia Chirurgica II[a], Universita degli Studi di Parma, Parma, Italy). *Chir. Patol. Sper.* 24(4):307-321; 1976.

0250 SPECIFIC INHIBITION OF MOUSE CELL PROLIFERATION BY AN AQUEOUS EXTRACT OF RABBIT COLON. (Fre.) Sassier, P.; Bergeron, M. (Dept. de physiologie, Universite de Montreal, Montreal, Quebec, Canada). *Clin. Res.* 24(5):666A; 1976.

0251 CHANGES IN THE GASTRIC MUCOSA IN DOGS FOLLOWING INTESTINAL RESECTION. (Spa.) Rodriguez Montes, J. A.; Spreafico, J. M.; Carmena, A.; Diaz Flores, L.; Arcelus, I. (Hospital Clinico "San Cecilio," Universidad de F Granada, Granada, Spain). *Rev. Esp. Enferm. Apar. Dig.* 48(6):673-682; 1976.

0252 QUANTITATIVE STUDY ON THE INFLUENCE OF DIFFERENT ALKALINE AGENTS ON THE EXPERIMENTAL ULCEROGENIC AND ANALGESIC ACTIVITY OF ASPIRIN. (Fre.) Hertz, F.; Lwoff, J. M.; Boissier, J. R. (Laboratoire de Pharmacologie II, UER Biomedicale des Cordeliers, 15, rue de l'Ecole-de-Medecine, F 75270 Paris Cedex 06, France). *Biol. Gastroenterol. (Paris)* 9(3):193-198; 1976.

0253 COMPARISON OF THE LEVEL OF THE INITIATED SERUM CHEMILUMINESCENCE AND THE BLOOD SERUM ENZYME ACTIVITIES IN DOGS WITH ACUTE HEPATIC INSUFFICIENCY. (Rus.) Evseev, N. G.; Farkhutdinov, R. R.; Mashkov, O. A.; Tishina, L. M. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Patol. Fiziol. Eksp. Ter.* (6):76-78; 1976.

0254 INTRACORPOREAL DIALYSIS IN HEPATIC INSUFFICIENCY. (Rus.) Ostroverkhov, G. E.; Berklavichus, V. Yu.; Bruslik, V. G. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Eksp. Khir. Anesteziol.* (4):41-45; 1976.

0255 SPECIFICITY OF β-GALACTOSIDASE IN HEPATOCELLULAR LESIONS. (Fre.) Smeesters, C.; Fourtanier, G.; Corman, J.; Daloze, P. (Dept. Chirurgie, Universite Montreal, Montreal, Canada). *Union Med. Can.* 105(10):1542-1544; 1976.

0256 INVAGINATION CHOLECYSTOSTOMY UNDER EXPERIMENTAL CONDITIONS. (Rus.) Margolis, G. A. (Izhevsk Medical Inst., Izhevsk, USSR). *Eksp. Khir. Anesteziol.* (4):64-65; 1976.

0257 ON THE SEQUELAE OF WEDGE-SHAPED RESECTION OF THE LIVER UNDER EXPERIMENTAL CONDITIONS. (Rus.) Toidze, Sh. S.; Izraelashvili, M. Sh. (Tbilisi Medical Inst., Tbilisi, USSR). *Eksp. Khir. Anesteziol.* (4):45-47; 1976.

0258 HOMOLOGOUS LIVER AND LIVER-KIDNEY TRANSPLANTATION. AN EXPERIMENTAL STUDY. (Ger.) Rigler, B. (Universitatsklinik fur Chirurgie Graz, Graz, Austria). *Z. Exp. Chir.* 9(5):262-281; 1976.

0259 REPAIR OF GASTRIC FUNDO-ANTRAL FISTULA BY HOMOLOGOUS AND HETEROLOGOUS FUNDO-ANTRAL TRANSPLANTATION IN THE ADULT RAT. (Fre.) Hung Le Van; Nin Khieu; Tuyet Nga Duong. (Laboratoire de Physiologie metabolique et Nutrition, Universite Pierre-et-Marie-Curie, 9, quai Saint-Bernard, 75005 Paris, France). *C. R. Acad. Sci. [D] (Paris)* 284(18):1823-1825; 1977.

0260 HEMIN COMPOUNDS OF BLOOD SERUM IN EXPERIMENTAL DESTRUCTIVE PANCREATITIS. (Rus.) Starosek, V. N.; Spitsyn, I. F. (Crimean Medical Inst., Simferopol, USSR). *Patol. Fiziol. Eksp. Ter.* (4):66-67; 1977.

0261 THE EFFICIENCY OF REVASCULARIZATION OF THE LIVER BY INTRAHEPATIC IMPLANTATION OF THE SPLENIC VASCULAR PEDICLE. (Fre.) Ionesco, G.; Neumann, E.; Lazar, A. (First Surgical Clinic, Str. Clinicilov 3, 3400 Cluj-Napoca, Rumania). *J. Chir. (Paris)* 112(3):199-208; 1976.

0262 ANTIBODIES TO A COUPLED MOLECULE OF SYNTHETIC GASTRIN IN THE BLOOD OF HEALTHY HUMANS. (Rus.) Polosatov, M. V. (Pavlov's Inst. Physiology, Acad. Science USSR, Leningrad, USSR). *Fiziol. Zh. SSSR* 63(5):750-752; 1977.

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0264 VARIATIONS IN THE PHAGOCYTOSIS CAPACITY OF KUPFER CELLS. (Rus.) Petrovitchev, N. N. (First Leningrad Medical Inst., Leningrad, USSR). *Tsitologiia* 18(12):1506-1507; 1976.

0265 TOTAL SYNTHESIS OF HUMAN BIG GASTRIN I AND THE 32-LEUCINE ANALOGUE: PRELIMINARY COMMUNICATION. (Ger.) Wunsch, E.; Wendlberger, G.; Hallett, A.; Jaeger, E.; Knof, S.; Moroder, L.; *et al.* (Max-Planck-Institut fur Biochemie, D-8033 Martinsreid bei Munchen, W. Germany). *Z. Naturforsch. [C]* 32(7/8):495-506; 1977.

0266 GENERAL ANESTHESIA IN THE RABBIT FOR SURGICAL RESEARCH ON THE DIGESTIVE TRACT.

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0267 ACUTE STRESS ULCER: EXPERIMENTAL STUDY ON ITS ETIOLOGY, PATHOPHYSIOLOGY, AND MANAGEMENT. (Jpn.) Yamaguchi, T. (Kyoto Univ. Sch. Medicine, Kyoto, Japan). *Arch. Jpn. Chir.* 46(3):278-308; 1977.

0268 CHANGES IN TISSUE ENERGY METABOLISM IN DEVELOPMENT OF EXPERIMENTAL ULCERS OF THE STOMACH. (Rus.) Anichkov, S. V.; Zavodskaya, I. S.; Moreva, E. V. (Inst. Experimental Medicine, Acad. Medical Science USSR, Leningrad, USSR). *Patol. Fiziol. Eksp. Ter.* (4):25-28; 1977.

0269 BODY ORIENTATION, FOOD DEPRIVATION AND POTENTIATION OF RESTRAINT-INDUCED GASTRIC LESIONS. (Eng.) Vincent, G.; Glavin, G.; Rutkowski, J.; Pare, W. (Veterans Admin. Hosp., Perry Point, MD 21902). *Gastroenterol. Clin. Biol.* 1(6/7):539-543; 1977.

0270 SERUM PROTEIN CHANGES IN EXPERIMENTAL INTESTINAL BYPASS. (Pol.) Pardela, M.; Rudnicki, M. (Acad. Medicine, Katowicach, Poland). *Pol. Przegl. Chir.* 49(6):593-597; 1977.

0271 EFFECT OF EXPERIMENTAL INTESTINAL BYPASS ON FUNCTIONAL AND SUBMICROSCOPIC CHANGES IN THE LIVER DEPENDING ON THE DURATION OF OBSERVATION. (Pol.) Pardela, M. (ul. PPr 6/4, 41-800 Zabrze, Poland). *Pol. Przegl. Chir.* 49(6):567-572; 1977.

0272 EFFECT OF ASPARTATE AND METHANDROSTENOLON IN LEAD-INDUCED LESIONS OF LIVER CELL ENZYMES. (Rum.) Barbarino, F.; Ghelberg, N. W.; Nagy, S. (Institutul de Igiena si Sanacate Publica, Cluj-Napoca, Rumania). *Rev. Ig.* [*Bacteriol.*] 26(2):129-136; 1977.

0273 FECAL FAT DETERMINATION IN HEALTHY CHILDREN. (Por.) Penna, F. J.; Roquete, M. L. V.; de Carvalho, A. S. T.; Mota, J. A. C.; Leao, E.; Ferreira, R. A.; *et al.* (Rua Silvestre Ferraz, 68--ap. 301, Sagrada Familia 30000, Belo Horizonte-MG, Brazil). *Arq. Gastroenterol.* 14(2):106-108; 1977.

0274 CHOICE OF THE OPTIMUM LEVEL OF RESECTION OF THE PANCREAS. (Rus.) Grigor'ev, V. A. (Kalinin Medical Inst., Kalinin, USSR). *Vestn. Khir.* 118(5):48-52; 1977.

0275 PARENTERAL FEEDING IN PATIENTS IN THE IMMEDIATE POSTOPERATIVE PERIOD AFTER OPERATIONS ON GASTROINTESTINAL TRACT. (Rus.) Lytkin, M. I.; Chaplygina, Z. A.; Shanin, S. S. (Inst. Advanced Training of Physicians, Leningrad, USSR). *Vestn. Khir.* 118(5):104-107; 1977.

0276 VIP IN HUMAN NEONATES AND INFANTS AS MEASURED BY RADIOIMMUNOASSAY AND RADIORECEPTOR ASSAY. (Fre.) Dupont, C.; Besson, J.; Laburthe, M.; Bataille, D.; Rosselin, G. (Hopital Saint-Antoine, 184, rue du Faubourg Saint-Antoine, F 75571 Paris Cedex 12, France). *Ann. Endocrinol. (Paris)* 38(2):151-152; 1977.

0277 MORPHOLOGICAL INVESTIGATIONS OF EXTRAHEPATIC DISORDERS AFTER PORTACAVAL END-TO-SIDE ANASTOMOSIS IN RATS. (Ger.) Rasenack, U.; Grun, H.; Liehr, H. (Medizinische Univ.-Klinik Wurzburg, Josef-Schneider-Str. 2, D-8700 Wurzburg, W. Germany). *Z. Gastroenterol.* 15(7):457-468; 1977.

0278 INTESTINAL ANASTOMOSIS BY MICROSURGICAL TECHNIQUE. (Ger.) Lamesch, A.; Dociu, N. (Kinderklinik St. Katharinen, Zurmaiener Strasse 9-11, 5500 Trier, W. Germany). *Z. Kinderchir.* 21(3):263-274; 1977.

0279 DISTURBANCES OF MICROCIRCULATION IN PERITONITIS AND ACUTE ILEUS IN THE ANGIOGRAPHIC PICTURE. (Ger.) Salkov, Ju. L.; Levendjuk, A. M. (Forschungsinstitut fur Allgemeine und Unfallchirurgie, Vjezd Balakireva 1, Charkow 18, USSR). *Radiol. Diagn. (Berl.)* 18(2):205-210; 1977.

0280 EFFECT OF THE PANCREATIC KALLIKREIN AND TRYPSIN INHIBITORS ON THE COURSE OF POSTISCHEMIC TOXEMIA. (Rus.) Kharchenko, V. Z.; Protsenko, V. A. (Kiev Inst. Pediatrics, Obstetrics, and Gynecology, Kiev, USSR). *Farmakol. Toksikol.* 40(5):606-613; 1977.

0281 VELOCITY OF INJECTION OF CONTRAST SUBSTANCE DURING AIMED ARTERIOGRAPHY IN ACUTE HEMORRHAGE INTO INTESTINAL TRACT. (Cze.) Vitek, J.; Potrusil, B.; Prochazka, J.; Kastak, B.; Krejcova, H. (Pekarska 53, 656 51 Brno, Czechoslovakia). *Cesk. Radiol.* 31(3):167-171; 1977.

0282 NEW KNOWLEDGE ON ARTERIAL BLOOD SUPPLY OF THE THIRD AND FOURTH PARTS OF THE DUODENUM: SURGICAL APPLICATIONS. (Fre.) Landier, J. F.; Calmat, A.; Honnart, F.; Clot, J. P.; Cabrol, C. (Pitie-Salpetriere, 105, boulevard de l'Hopital, 75013 Paris, France). *J. Chir. (Paris)* 113(4):405-411; 1977.

See also, 0010, 0020, 0098, 0223, 0718.

DIAGNOSTIC PROCEDURES

0283 ULTRASONIC DISTINCTION OF ABSCESSES FROM OTHER INTRA-ABDOMINAL FLUID COLLECTIONS. (Eng.) Doust, B. D.; Quiroz, F.; Stewart, J. M. (Medical Coll. Wisconsin, 8700 W. Wisconsin Ave., Milwaukee, WI 53226). *Radiology* 125(1):213-218; 1977.

To investigate the ultrasonic distinction of abscesses from other intra-abdominal fluid collections, the nature of 103 intra-abdominal fluid collections examined by ultrasound was determined by autopsy, needle aspiration, spontaneous drainage, or surgical exploration. Abscesses, chronic hematomas, lymphoceles, urinomas, cysts, and fluid-filled bowel loops were all approximately elliptical or circular in cross section. Collections of ascites had a very irregular outline. Bowel loops appeared as multiple circles, all of similar size. Chronic hematomas contained strongly echoing material, while lymphoceles, urinomas, renal and hepatic cysts, and collections of ascites were echo-free. One ovarian cyst into which there had been bleeding contained echoes. The walls of abscesses, chronic hematomas, and bowel loops were less sharp than the walls of other collections. Intrahepatic abscesses had more ragged walls than abscesses elsewhere. Ultrasonic characteristics of abscesses and hematomas overlapped. It is sometimes possible to distinguish the nature of the fluid by its ultrasonic properties, but atypical appearances are not rare.

0284 HEPTADECAPEPTIDE GASTRIN: MEASUREMENT IN BLOOD BY SPECIFIC RADIOIMMUNOASSAY. (Eng.) Dockray, G. J.; Taylor, I. L. (Dept. Physiology, Univ. Liverpool, Brownlow Hill, P.O. Box 147, Liverpool L69 3BX, England). *Gastroenterology* 71(6):971-977; 1976.

The characteristics of an antibody (designated L6) that has absolute specificity for heptadecapeptide gastrin are described. This antibody was found to bind G17, but not peptide fragments or molecular forms of gastrin comprising G17 with either amino acid deletions, or additions, at the carboxyl- and amino-terminals. In sera from patients with Zollinger-Ellison syndrome, the only form of gastrin revealed by L6 was compatible with G17, and there was good agreement between estimated G17 concentrations in serum analyzed by gel filtration and by direct radioimmunoassay using L6. Using L6 in conjunction with antibodies specific for carboxyl- and amino-terminals of G17, it was possible to measure concentrations of different forms of gastrin in the sera of normal subjects after a meal in greater detail than was previously possible. After a light meal consisting of eggs, toast, and Oxo, serum concentrations of G17 measured by L6 increased to a peak 20 min after feeding (Δ gastrin, 19 pM; n=17). In contrast, the concentration of G34 peaked at 50 min (Δ gastrin, 27 pM). Small amounts of amino-terminal fragments of G17 were present throughout the digestive period. Applying the known ratio of biological potencies of G34 and G17 for stimulation of acid secretion in man, it is estimated that G17 accounts for about 75% of the biological activity in blood after a meal, even though G34 is present in higher molar concentrations.

0285 LYSOZYME LEVELS IN CHRONIC INFLAMMATORY BOWEL DISEASES. (Ger.) Rollinghoff, W.; Brandes, J. W.; Ehms, H.; Miller, B.; Schmulling, R. M.; Tischendorf, F. W.; Malchow, H. (Abt. Innere Medizin I, Otfried-Muller-Strasse, D-7400 Tubingen, W. Germany). *Klin. Wochenschr.* 55(5):225-230; 1977.

Urinary and serum lysozyme concentrations were determined by the lysoplate method of Osserman in 101 patients with Crohn's disease and 26 patients with ulcerative colitis and compared to 38 normal controls. Of the 101 patients with Crohn's disease, 29 showed changes of the small intestine, 31 had involvement of the small and large colon, and 16 showed involvement of the colon, 5 of the duodenum, and 1 of the stomach. At the time of the study, 14 patients were treated with prednisolone, 18 with salazopyrine, 23 with both the preceding agents, and 7 azathioprine; 28 had no therapy. Of the 26 patients with ulcerative colitis, the colon was extirpated in 3, 20 received salazopyrine, 1 received prednisolone, 4 received both of the latter 2 drugs, and 4 had no therapy. The mean serum lysozyme level of the patients with Crohn's disease was 8.2 ± 2.6 μg/ml as compared with 8.6 ± 2.9 μg/ml for the patients with ulcerative colitis and 8.4 ± 1.8 μg/ml for controls; no significant differences were noted between the patients and controls. Furthermore, no correlation between serum lysozyme levels and the disease activity index was found. However, serum lysozyme levels were significantly higher in patients with Crohn's disease of the small and large bowel (9.5 ± 3.5 μg/ml) than in patients with involvement of the small bowel only (8.1 ± 2.4 μg/ml) or operated patients (7.5 ± 1.5 μg/ml). The mean urinary lysozyme levels in 74 patients with Crohn's disease was 0.9 ± 2.1 μg/ml (normal value 0.9 ± 0.9 μg/ml) as compared with 0.1-16.6 μg/ml for 26 patients with ulcerative colitis. The results suggest that the determination of serum and urinary lysozyme levels is unsuitable for the differential diagnosis of inflammatory bowel disease and for assessment of disease activity.

0286 CHANGES IN THE ARCHITECTONICS OF AZYGOUS VISCERAL ARTERIES AFTER SURGICAL INTERVENTIONS ON ABDOMINAL ORGANS. (Rus.) Prokubovsky, V. I.; Efleev, V. P.; Frantsev, Ju. V. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Vestn. Khir.* 118(2):51-56; 1977.

0287 RETROGRADE CATHETERIZATION OF THE UMBILICAL VEIN: MANOMETRIC AND RADIOLOGIC DATA. (Spa.) Soria, T.; Parrilla, P.; Benages, A.; Almenar, L.; Mora, F.; Molina, R.; *et al.* (Hospital Clinico Universitario, Valencia, Spain). *Rev. Esp. Enferm. Apar. Dig.* 48(3):301-318; 1976.

0288 RESULTS WITH DEFEROXAMINE IN THE DIAGNOSIS OF HYPO-, NORMO- AND HYPERSIDEROTIC HYPO-

SIDEREMIA. (Ita.) Di Lollo, F.; Morini, P. L.; Fazzini, G.; Niosi, L. (S. Maria Nuova Hosp., Florence, Italy). *Minerva Med.* 68(9):559-567; 1977.

0289 METHODS FOR EVALUATING THE ABSORPTION OF CALCIUM IN THE HUMAN DIGESTIVE TRACT. (Ita.) Gennari, C. (Cattedra di Medicina Nucleare, Universita degli Studi, Siena, Italy). *Minerva Med.* 68(15):943-950; 1977.

0290 STABLE ISOTOPES IN GASTROENTEROLOGY. (Rus.) Belousov, A. S.; Bredikhina, N. A.; Zhuravlev, L. T. (Central Inst. Advanced Medical Studies, Moscow, USSR). *Sov. Med.* (2):13-16; 1977.

0291 CURRENT STATUS OF PROXIMAL DIGESTIVE ENDOSCOPY. (Fre.) Savary, M. (Centre Hospitalier Universitaire Vaudois, 1000 Lausanne, Switzerland). *Rev. Fr. Gastroenterol.* 127(7F):51-56; 1977.

0292 CUMULATION OF ENDOSCOPIC FINDINGS. (Ger.) v. Gaisberg, U.; Heinkel, K.; Kimmig, J. M. (Med. Klinik Stuttgart-Bad Cannstadt, Priessnitzweg 24, D-7000 Stuttgart 50, W. Germany). *Z. Gastroenterol.* 14(7):706-708; 1976.

0293 ROENTGENOLOGICAL PRACTICE. (Fre.) Faure, C.; Montagne, J. Ph. (Hopital Trousseau, 8-28, avenue Arnold-Netter, 75571 Paris Cedex 12, France). *Arch. Fr. Pediatr.* 34(2):193-194; 1977.

0294 RADIOLOGICAL DIAGNOSIS OF CHANGES IN THE UPPER DIGESTIVE TRACT. STRATEGY AND TECHNIQUE. (Ger.) Ordnung, W. (Heinz-Kalk-Klinik, Am Gradierbau, D-8730 Bad Kissingen, W. Germany). *Med. Klin.* 72(12):524-529; 1977.

0295 PANENDOSCOPY: INDICATION VS. FINDINGS. (Ger.) Jenny, S.; Kobler, E.; Nuesch, H. J.; Deyhle, P. (Medizinische Universitatspoliklinik, Departement fur Innere Medizin, Ramistrasse 100, Ch-8091 Zurich, Switzerland). *Z. Gastroenterol.* 14(7):708-709; 1976.

0296 MEASUREMENT OF OCCULT GASTROINTESTINAL HEMORRHAGE BY ^{51}Cr TEST DURING LONG-TERM ANTI-INFLAMMATORY TREATMENT. (Fre.) Tamisier, J. N.; Serot, J. M.; Netter, P.; Robert, J.; Pourel, J.; Gaucher, A. (Clinique Rhumatologique, Nancy, France). *Rev. Fr. Gastroenterol.* (122):37-45; 1976.

0297 IS IT POSSIBLE TO IMPROVE THE PROGNOSIS OF UPPER DIGESTIVE TRACT HEMORRHAGES (OTHER THAN PORTAL HYPERTENSION)? (Fre.) Pascal, J. P.; Tournut, R. (Service de Gastro-Enterologie, C.H.U. Toulouse-Purpan, F 31052 Toulouse Cedex, France). *Gastroenterol. Clin. Biol.* 1(5):409-414; 1977.

0298 INTERPRETATION OF THE RADIOLOGICAL PICTURE OF THE UPPER SEGMENT OF THE SMALL INTESTINE IN CHILDREN. (Fre.) Montagne, J. Ph.; Faure, Cl. (Hopital Trousseau, 8-28, avenue Arnold Netter, 75571 Paris Cedex 12, France). *Arch. Fr. Pediatr.* 34(1):90-104; 1977.

0299 A MODIFIED GUAIAC TEST FOR THE EARLY DETECTION OF TUMORS OF THE INTESTINAL TRACT. (Ger.) Warm, K.; Blazek, Z.; Weithofer, G.; Bloch, R. (Klinik fur Verdauungs- und Stoffwechselkrankheiten, Am Weinberg 3-5, D-6430 Bad Hersfeld, W. Germany). *Munch. Med. Wochenschr.* 119(9):285-288; 1977.

0300 ERRORS IN THE DIAGNOSIS OF INTESTINAL LIPOMA. (Rus.) Makhov, N. I. (Moscow Medical Inst. Stomatology, Moscow, USSR). *Khirurgiia (Mosk.)* (2):114-117; 1977.

0301 (D+) XYLOSE TEST. (Rus.) Alimova, M. M. (Inst. Pediatrics, Acad. Medical Sciences, Moscow, USSR). *Pediatriia* (7):82-85; 1976.

0302 MALABSORPTION OF BILE ACIDS IN CHILDREN WHO DID NOT UNDERGO INTESTINAL RESECTION. (Fre.) Weber, A.; Roy, Cl. C.; Chartrand, L.; Morin, Cl. L.; Van Caillie, M. (Conseil de la Recherche Medicale du Quebec, Canada). *Rev. Fr. Gastroenterol.* (122):25-31; 1976.

0303 SIMPLIFIED HYPOTONIC DUODENOGRAPHY AND IMPROVED DOUBLE-CONTRAST VISUALIZATION OF THE STOMACH. (Ger.) Gerstner, H. D. (Radiologische Klinik, Stadt. Krankenanstalten, 6600 Saarbrucken 6, Winterberg, W. Germany). *Med. Welt* 27(44):2106-2109; 1976.

0304 EVALUATION OF COLONIC PATHOLOGIES: COLONOSCOPY AND RADIOLOGY. (Fre.) Archambault, A.; Bourgie, J.; Lamoureux, C. (Hopital Maisonneuve-Rosemont, Montreal, Canada). *Union Med. Can.* 106(6):853-861; 1977.

0305 COMPARATIVE PHARMACODYNAMIC COLON DOUBLE CONTRAST EXAMINATION WITH GLUCAGON AND ATROPINE. (Ger.) Poser, H.; Baier, J. (Chirurgischen Universitatsklinik, Hospitalstrasse 40, D-2300 Kiel, W. Germany). *Radiol. Diagn.* 18(3):355-358; 1977.

0306 INDIRECT SIGNS OF GASTRIC AND DUODENAL DISEASES AS EVIDENCED BY FIBROGASTROSCOPY.

(Rus.) Chekanov, B. Ya.; Kats, V. N. (Saratov Medical Inst., Saratov, USSR). *Vestn. Khir.* 118(4): 39-43; 1977.

0307 RADIOGRAPHIC INVESTIGATION OF THE STOMACH WITH DOUBLE CONTRAST. (Dan.) Thogersen, G. (Slotsparken 24, DK-2880 Bagsvaerd, Denmark). *Ugeskr. Laeger* 138(41):2492-2493; 1976.

0308 OPTIMAL SIMULTANEOUS EXAMINATION OF THE MAIN GASTRIC FUNCTIONS. (Rus.) Lukash, N. V.; Skripka, V. I. (Krymsk Medical Inst., Krymsk, USSR). *Vrach. Delo* (2):98-100; 1977.

See also, 0016, 0064, 0066, 0093, 0099, 0239, 0273, 0279, 0386, 0310, 0315, 0327, 0328, 0332, 0340, 0344, 0361, 0362, 0389, 0399, 0436, 0447, 0448, 0449, 0457, 0467, 0485, 0490, 0498, 0504, 0506, 0514, 0515, 0516, 0517, 0519, 0529, 0534, 0542, 0560, 0562, 0568, 0576, 0599, 0616, 0624, 0628, 0635, 0662, 0663, 0674, 0688, 0689, 0698, 0699, 0702, 0707, 0708, 0709, 0741, 0746, 0749.

GASTROINTESTINAL DISEASES

ESOPHAGUS

0309 RESPONSE OF LOWER ESOPHAGEAL SPHINCTER PRESSURE TO BEEF SOUP OR AOC-TETRAPEPTIDE STIMULATION IN ESOPHAGITIS. (Eng.) Nagasaki, A.; Ishimori, A.; Masamune, O.; Yamagata, S. (Tohoku Univ. Sch. Medicine, Sendai, Japan). *Tohoku J. Exp. Med.* 121(1):91-97; 1977.

Lower esophageal sphincter (LES) pressure in the resting state and after stimulation with beef soup or anodal opening contraction (AOC) tetrapeptide were measured in healthy subjects and diseased patients by an infused open-tip method. The mean basal LES pressures in healthy subjects and in patients with gastric ulcer, duodenal ulcer, scleroderma without gastrointestinal symptoms, and esophageal hiatal hernia without esophagitis were 13.1 ± 1.7, 12.8 ± 1.4, 13.7 ± 1.4, 12.0, and 12.8 ± 1.4 cm water, resp. (differences not significant). In two cases of Zollinger-Ellison syndrome, the resting LES pressure was 24.1 ± 0.5 cm, which was significantly ($p<0.001$) higher than that in controls. In cases of esophagitis with hiatal hernia, esophagitis without hiatal hernia, and esophagitis following Billroth II gastrectomy, the LES pressure levels were 10.2 ± 1.2 ($0.001<p<0.01$), 10.1 ± 0.9 ($0.001<p<0.01$), and 6.9 ± 1.2 cm ($p<0.001$), resp., significantly lower than in controls. In cases of esophagitis, the resting LES pressure and corresponding fasting plasma gastrin level tended to be lower than those in healthy subjects, but the reverse was true in cases of Zollinger-Ellison syndrome. When 200 ml beef soup was infused into the stomach, the peak LES pressures of healthy subjects and patients with esophagitis were 25.7 ± 0.5 and 17.1 ± 1.8 cm, resp. The peak percentage response to the resting LES pressure in these two groups was 221.0 ± 4.2 and 180.5 ± 10.5, resp. This implies that the peak LES pressure and the peak percentage response in the LES pressure were lower in esophagitis patients than in healthy subjects. After the i.v. administration of 0.3 or 0.5 μg/kg of tetrapeptide, these parameters were again lower in esophagitis patients than in controls. It is concluded that there are some factors other than gastrin that make the LES pressure lower in esophagitis patients than in controls.

0310 GASTRO-OESOPHAGEAL SPHINCTER PRESSURE, MOTILITY AND ACID CLEARING. A STUDY OF HIATUS HERNIA PATIENTS AND NORMAL SUBJECTS, AND OF THE EFFECT OF A MODIFIED BELSEY MK IV REPAIR ON THE RESULTS OF THE MANOMETRIC AND ACID-CLEARING TESTS. (Eng.) Boesby, S. (Odense Sygehus, DK-Odense, Denmark). *Scand. J. Gastroenterol.* 12(4): 407-416; 1977.

The results of manometric and acid-clearing studies of patients with hiatal hernia were compared with those of normal subjects, and the effect of a modified Belsey MK IV repair for hiatal hernia on the results of the manometric and acid-clearing studies was evaluated. Basal gastroesophageal sphincter pressure was recorded in 68 patients with symptomatic, radiologically verified sliding hiatal hernia, and in 37 healthy subjects. The diameter of the probe was 2.5 mm, and the flow rate 0.5 ml/min. Mean sphincter pressure was lower in patients (6 mm Hg) than in normal subjects (15 mm Hg). In 32% of the patients, the sphincter pressure was within normal range (8-24 mm Hg), whereas in 68% it was lower than in the normal subjects. There seemed to be no relationship between sphincter pressure and severity of symptoms. Esophageal acid clearing was investigated in 57 of the patients and in 26 of the normal subjects. Normal subjects cleared the bolus of acid in an average of 10 swallows (range 4-16). In patients, the incidence of prolonged clearing was greater, but the acid-clearing ability did not seem to be related to the degree of severity of the symptoms. A manometric study was carried out in 45 patients and an acid-clearing study was carried out in 40 patients, before and 3 months after a modified Belsey MK IV repair. The mean postoperative sphincter pressure was higher (10 mm Hg) than the preoperative mean (6 mm Hg), but was still lower than the normal mean. Significant changes in acid-clearing ability were not demonstrated. The results are inconclusive with regard to the importance of esophageal motility disturbances for the symptomatology and acid-clearing ability. Thirty-seven of 45 patients became asymptomatic, and all but one of the remaining patients improved.

0311 FUNDOPLICATION FOR THE TREATMENT OF GASTRO-ESOPHAGEAL REFLUX IN HIATAL HERNIA. (Eng.) Rossetti, M.; Hell, K. (Kantonsspital, CH-4410 Liestal, Switzerland). *World J. Surg.* 1(4):439-444; 1977.

Experience with 1,400 patients who underwent fundoplication for the treatment of reflux in hiatal hernia was reviewed to determine the correct indications for surgery. These indications include (a) a retrosternal burning sensation (in 90% of cases), (b) objective confirmation of reflux by means of x-ray and endoscopic examination, together with biopsy examination of the esophageal mucosa and gastric acid evaluation, and (c) evidence of organic complications, such as endobrachyesophagus with ulcerostenotic changes at the junction between the esophageal and gastric mucosa. Long-term follow-up of 590 patients with simple reflux esophagitis who underwent fundoplication showed that 87.5% were symptom free. In 44 patients with complicated gastroesophageal reflux disease, fundoplication produced clinical healing in 84.1%.

0312 CONSIDERATIONS ON A CASE OF CORK-SCREW ESOPHAGUS. (Ita.) Valentino, M. L.; Sansone, D. (Ospedale Riuniti di Napoli, Naples, Italy). *Rass. Int. Clin. Ter.* 57(9):626-631; 1977.

0313 A CASE REPORT OF AN ESOPHAGEAL FIBROLIPOMA. (Ger.) Merck, W.; Weerda, H. (Universitats-HNO-Klinik, Killianstr. 5, D-7800 Freiburg/Br., W. Germany). *HNO* 25(6):204-205; 1977.

0314 ESOPHAGEAL CANCER MORBIDITY AS EVIDENCED BY THE GENEALOGY OF PATIENTS REGISTERED IN THE GUR'EV PROVINCE. (Rus.) Nasipov, S. N. (Gur'ev Regional Oncology Clinic, USSR). *Vopr. Onkol.* 23(8):81-85; 1977.

0315 OESOPHAGOSCOPY. (Eng.) Bennett, J. R. *In: Modern Topics in Gastrointestinal Endoscopy.* Edited by K. F. R. Schiller and P. R. Salmon. (Chicago: William Heinemann Medical Books): 83-97; 1976.

0316 HAEMATEMESIS AND MELAENA. (Eng.) Chandler, G. N.; Walls, W. D.; Glanville, J. N. *In: Modern Topics in Gastrointestinal Endoscopy.* Edited by K. F. R. Schiller and P. R. Salmon. (Chicago: William Heinemann Medical Books): 109-116; 1976.

0317 MOTOR DISORDERS OF THE ESOPHAGUS. (Eng.) Henderson, R. D. (Baltimore: Williams & Wilkins): 231 pp.; 1976.

0318 FEEDING OF PATIENTS WITH ESOPHAGEAL INJURY. (Rus.) Abakumov, M. M.; Burdyga, F. A. (N. V. Sklifosovsky Scientific Res. Inst. First Aid, Moscow, USSR). *Vestn. Khir.* 118(5):24-27; 1977.

0319 REFLUX ESOPHAGITIS AND STRICTURE OF THE ANASTOMOSIS AFTER GASTRECTOMY. (Rus.) Britvin, A. A. (Vologodsy Regional Oncology Clinic, USSR). *Khirurgiia (Mosk.)* (8):21-24; 1977.

0320 PREVENTION OF ESOPHAGEAL INJURIES DURING BOUGINAGE. (Rus.) Kanshin, N. N.; Abakumov, M. M.; Sapozhnikova, M. A. (N. V. Sklifosovsky Scientific Res. Inst. First Aid, Moscow, USSR). *Khirurgiia (Mosk.)* (6):8-12; 1977.

0321 RESULTS OF THE SURGICAL TREATMENT OF HIATAL HERNIA IN THE ADULT USING A MODIFIED ALLISON TECHNIQUE: REPORT OF 82 CASES. (Fre.) Skalkeas, G. D. (St. Paul Univ. Hosp., Athens, Greece). *Chirurgie* 103(6):419-423; 1977.

0322 THERAPY OF ESOPHAGEAL REFLUX. (Ger.) Ottenjann, R. (Stadt. Krankenhaus Munchen-Neuperlach, Oskar Maria Graf-Ring 51, 8 Munchen 83, W. Germany). *Fortschr. Med.* 95(34):2098-2101; 1977.

0323 ESOPHAGO-AORTAL FISTULA AS A COMPLICATION OF TRACTION DIVERTICULUM. (Ger.) Margreiter, R.; Suckert, K. (Chirurgische Univ.-Klinik, Anichstrasse 35, A-6020 Innsbruck, Austria). *Chirurg* 48(5):331-333; 1977.

0324 FAILURES AND SUGGESTED FAILURES OF HELLER'S OPERATION FOR IDIOPATHIC MEGA-ESOPHAGUS: STUDY OF 55 REOPERATED CASES. (Fre.) Fekete, F.; Lortat-Jacob, J. L. (Hopital Beaujon, 100, boulevard du General Leclerc, 92110 Clichy, France). *Ann. Chir.* 31(6):515-524; 1977.

0325 LONG-TERM PROSPECTIVE FOLLOW-UP OF CHILDREN WITH A HIATAL HERNIA (PARTIAL THORACIC STOMACH) [Abstract]. (Eng.) Carre, I. J.; Astley, R.; Langmead-Smith, R. (Dept. Child Health, Queen's Univ. Belfast, Belfast, Northern Ireland). *Gut* 18(11):A990; 1977.

0326 THE TRANSJUGULAR TECHNIQUE FOR HEPATIC VENOGRAPHY AND BIOPSY CHOLANGIOGRAPHY AND OBLITERATION OF ESOPHAGEAL VARICES [Abstract]. (Eng.) Goldman, M. L.; Gonzalez, A. C.; Galambos, J. T.; Gordon, I.; Kibble, M. (Emory Univ. Affiliated Hosp., Atlanta, GA). *Gastroenterology* 73(5):1222; 1977.

0327 ENDOSCOPIC DIAGNOSIS AND THERAPY OF GASTROINTESTINAL HEMORRHAGE [Abstract]. (Eng.) Kiefhaber, P.; Nath, G.; Moritz, K.; Gorisch, W.; Kreitmair, A. (II. Med. Klinik, Ziemssenstr. 1, 8000 Munchen, W. Germany). *Endoscopy* 9(3):184-185; 1977.

0328 THE IMPORTANCE OF ENDOSCOPY IN REFLUX ESOPHAGITIS [Abstract]. (Eng.) Siewert, R. (Surgical Clinic, Univ. Gottingen, Gottingen, W. Germany). *Endoscopy* 9(3):185-186; 1977.

0329 REFLUX ESOPHAGITIS [Abstract]. (Eng.) Ottenjann, R. (Stadt. Krankenhauses Munchen-Neuperlach, Munchen, W. Germany). *Endoscopy* 9(3):185; 1977.

0330 RECURRENT BRONCHOPNEUMOPATHY DUE TO GASTRO-ESOPHAGEAL REFLUX: AN UNRECOGNIZED ENTITY. (Fre.) Dutau, G.; Terral Mias, A.; Ribot, C. (C.H.U. Rangueil, Toulouse, France). *Rev. Med. Toulouse* 12 (4):345-360; 1976.

0331 EFFECT OF NISSEN FUNDOPLICATION IN PATIENTS WITH GASTROESOPHAGEAL REFLUX [Abstract]. (Eng.) Bjerkeset, T.; Nordgard, K.; Schjonsby, H. (Haukeland Hosp., Bergen, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):9; 1977.

0332 DIAGNOSIS AND SURGICAL MANAGEMENT OF BENIGN DISEASES OF THE ESOPHAGUS. (Eng.) Nemir, P., Jr.; Wallace, H. W.; Fallahnejad, M.

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(Faculty Medicine, Univ. Pennsylvania, Philadelphia, PA). *Curr. Probl. Surg.* 13(3):1-74; 1976.

0333 ESOPHAGEAL ATRESIA AND ASSOCIATED ANOMALIES. (Eng.) German, J. C.; Mahour, G. H.; Woolley, M. M. (Children's Hosp., Los Angeles, CA). *J. Pediatr. Surg.* 11(3):299-306; 1976.

0334 ESOPHAGEAL CARCINOMA WITH PSEUDOHYPERPARATHYROIDISM AND HYPERCORTICISM. (Eng.) Kumar, G. K.; Naidu, V. G.; Razzaque, M. A. (Wayne State Univ. Sch. Medicine, Detroit, MI). *Am. J. Gastroenterol.* 65(3):222-225; 1976.

See also, 0292, 0295, 0297, 0353, 0361, 0443, 0660, 0673.

STOMACH

0335 IMMUNOHISTOCHEMICAL INVESTIGATION OF GASTRIN-PRODUCING CELLS (G CELLS): THE DISTRIBUTION OF G CELLS IN RESECTED HUMAN STOMACHS. (Eng.) Stave, R.; Brandtzaeg, P. (Rikshospitalet, Oslo, Norway). *Scand. J. Gastroenterol.* 11(7):705-712; 1976.

Direct immunofluorescence with rhodamine-labeled rabbit antigastrin IgG conjugate was used to systematically map the G-cell distribution in resected stomachs from eight gastric ulcer patients and eight duodenal ulcer patients with uremia. G cells were not found in the body of the stomachs. A low number of G cells, which amounted to about 10 G cells per mucosal unit (MU, 6 μm thick and 1 mm wide block of tissue from the muscularis mucosae to the surface epithelium) occurred in a transitional zone at the proximal border of the antrum in both gastric ulcer and duodenal ulcer patients. No significant difference was found between the number of G cells per mucosal unit at various levels of the antrum in gastric ulcer patients; G cells/mucosal unit ranged from about 40-50 in these cases. In duodenal ulcer patients with uremia, there was a significant increase in the G cell number from the proximal (about 40 cells/MU) to the middle antrum (about 55 cells/MU, $0.02<p<0.05$) and especially from the proximal to the distal antrum (about 80 cells/MU) ($p<0.01$). There was no significant difference in the circumferential distribution of antral G cells between the two groups of patients. Statistically significant differences ($0.02<p<0.05$) between the two patient groups appeared for G cell counts in the distal third part of the antrum, with the number of G cells/MU being about 50 for gastric ulcer patients and about 80 for duodenal ulcer patients. The proximal end of the duodenum contained considerably fewer G cells (about 10 cells/MU in both patient groups) than the antrum. The results confirm the reported preponderance of G cells in the middle and basal third of the antral mucosa, although no conclusions are made concerning the difference in antral G cell distribution in the two patient groups.

0336 MENETRIER'S DISEASE: EFFECT OF MEDICAL AND SURGICAL VAGOTOMY. (Eng.) Russell, I. J.; Smith, J.; Dozois, R. R.; Wahner, H. W.; Bartholomew, L. G. (Mayo Graduate Sch. Medicine, Univ. Minnesota, Rochester, MN). *Mayo Clin. Proc.* 52(2):91-96; 1977.

The case of a 62-yr-old woman with incapacitating atherosclerotic vascular occlusive disease who was found to have severe hypoproteinemia as a result of Menetrier's disease is described. That diagnosis was suspected on roentgenographic and gastroscopic examinations and confirmed by examination of full-thickness surgical gastric biopsy specimens. The protein loss from the stomach was significantly decreased by treatment with trimethaphan (0.3-0.5 mg/min, 4.5 hr, i.v.) and atropine (0.5 mg every 2 hr, i.m.), and a proximal gastric vagotomy was carried out at the time of endarterectomy. Subsequent protein turnover studies suggested that there was a significant decrease in protein loss by the stomach; plasma radioactive albumin clearance was 90 ml/day immediately after surgery and 77 and 33 ml/day at 4 and 12 months postoperatively, resp., and gastric clearance was 41 ml/24 hr at 12 months after surgery. Further evaluation of the possible role of medical and surgical vagotomy in this disease seems warranted.

0337 GASTRIC MUCOSAL POLYPS IN PERNICIOUS ANAEMIA. (Eng.) Elsborg, L.; Andersen, D.; Myhre-Jensen, O.; Bastrup-Madsen, P. (Aarhus Amtssygehus, DK-8000 Aarhus C, Denmark). *Scand. J. Gastroenterol.* 12(1):49-52; 1977.

Sixty-eight patients with pernicious anemia were followed up for 4 yr to determine whether gastric mucosal polyps were present. Gastric polyps were observed in 25 patients. In nine patients the polyps completely vanished in a year or two. The number of polyps decreased during the observation

period in two patients. New polyps developed in five patients in whom gastroscopy had showed none. None of these polyps underwent malignant transformation during the period of observation, and by histological examination they were classified as non-neoplastic "regenerative" benign polyps in an atrophic gastric mucosa. The surgical attitude to these non-neoplastic gastric polyps in pernicious anemia should be restrictive.

0338 ENDOSCOPIC FINDINGS OF BENIGN AND MALIGNANT SMOOTH MUSCLE TUMOURS OF THE STOMACH AND DUODENUM [Abstract]. (Eng.) Hradsky, M.; Furugard, K.; Gad, A.; Willen, R. (Falun Hosp., Falun, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):36; 1977.

0339 VARIABILITY OF GASTRITIS AND ATROPHY IN INDIVIDUAL HUMAN STOMACHS [Abstract]. (Eng.) Elgjo, K.; Stave, R.; Brandtzaeg, P.; Nygaard, K. (Natl. Hosp. Norway, Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):18; 1977.

0340 DIAGNOSIS OF ADENOCARCINOMA OF THE STOMACH: A CORRELATION BETWEEN BRUSH CYTOLOGY AND ENDOSCOPIC BIOPSY WITH REFERENCE TO GASTROSCOPIC AND ROENTGENOLOGIC INVESTIGATIONS [Abstract]. (Eng.) Willen, R.; Gad, A.; Willen, H.; Hradsky, M.; Furugard, K. (Falun Hosp., Falun, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):114; 1977.

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See also, 0085, 0100, 0101, 0104, 0231, 0267, 0268, 0269, 0284, 0292, 0297, 0303, 0306, 0307, 0310, 0311, 0316, 0319, 0322, 0325, 0327, 0330, 0331, 0367, 0369, 0370, 0371, 0373, 0374, 0380, 0385, 0387, 0414, 0510, 0696, 0709, 0724, 0737.

STOMACH

Peptic Ulcers

0363 BASAL GASTRIC SECRETION: REPRODUCIBILITY AND RELATIONSHIP WITH DUODENAL ULCERATION. (Eng.) Faber, R. G.; Hobsley, M. (Middlesex Hosp. Medical Sch., London, England). *Gut* 18(1):57-63; 1977.

Basal gastric secretion was examined in control subjects (C), duodenal ulcer patients (DU), and DU patients after vagotomy (DU + V, with or without a drainage procedure). Secretion in terms of observed gastric juice volume (Vo) and of gastric juice volume corrected for pyloric losses and duodenal reflux (Vg) was significantly ($p<0.01$) less in the second than in the first half-hour of a 1-hr basal collection; C, DU, and DU + V groups showed Vo values of 4.387, 2.739, and 4.914 ml/hr, resp., and Vg values of 7.537, 7.242, and 4.353 ml/hr, resp. Basal acid output (BAO) was significantly ($p<0.05$) less also in the second half-hour of a 1-hr basal collection in controls and DU + V patients; the BAO values for C, DU, and DU + V groups were 2.102, 1.639, and 2.863, mM/hr, resp. The reproducibility of the second half-hour basal collections taken on two different occasions from DU and DU + V patients was poor; the 95% tolerance limits of the difference between the two tests in terms of Vo, titratable acidity (H^+), BAO, and VG were approximately 101.2 ml/hr, 42.2 mM, 9.28 mM/hr, and 107.6 ml/hr, resp., for DU patients and 106.0 ml/hr, 46.0 mM, 7.28 mM/hr, and 93.8 ml/hr, resp., for DU + V patients. When expressed in terms of basal secretion in excess of the upper 95% tolerance limits of C subjects, the percentages of DU patients exceeding these limits were 17 for Vo, 0 for H^+, 29 for BAO, and 19 for Vg. The percentages of DU + V patients with recurrent ulcer after vagotomy exceeding the 95% tolerance limits of DU + V patients without symptoms were 25 for Vo, 0 for H^+, 50 for BAO, and 25 for Vg. By all indices of secretion, basal gastric secretion in DU patients was significantly ($p<0.005$) greater than in C subjects or in asymptomatic DU + V patients. Secretion by all indices was also significantly ($p<0.05$) greater in recurrent ulcer DU + V patients than in asymptomatic DU + V patients. When changes in basal secretion were examined before and after vagotomy, only in terms of Vg was there a significant ($p<0.01$) difference between the two groups. The overlap in the results from different groups of patients was so great

in general that basal secretion could not be ascribed an etiological role in the majority of patients with duodenal ulcer.

0364 THE ROLE OF THE BETA-ADRENERGIC RECEPTOR IN THE SECRETION OF GASTRIN: STUDIES IN NORMAL SUBJECTS AND IN PATIENTS WITH DUODENAL ULCERS. (Eng.) Brandsborg, O.; Brandsborg, M.; Christensen, N. J. (Kommunehospitalet, Aarhus C, Denmark). *Eur. J. Clin. Invest.* 6(5):395-401; 1976.

The role of beta-adrenergic receptors in gastrin secretion was studied in 8 normal men and 11 patients with duodenal ulcer. The i.v. infusion of isoproterenol (2 μg/min, 20 min) increased the serum gastrin concentration more in the patients with a duodenal ulcer than in the healthy subjects, and the differences were significant whether the absolute values were compared (mean rise 12 ng/l in controls versus 82 ng/l after isoproterenol, $p<0.01$) or the increase in serum gastrin expressed as a percent of the basal concentration was used (52% versus 94%, $p<0.05$). The rise in pulse rate, blood glucose concentration, and serum insulin was the same in both groups. Gastrin secretion was also increased significantly more in the patients than in the control subjects after a beef meal. Basal serum gastrin concentrations were higher in the patients than in the control subjects and correlated to the rise in serum gastrin during both tests in the duodenal ulcer patients. Isoproterenol- and meal-stimulated gastrin secretion, expressed as percent of the basal value, was twice as high in the patients as in the control subjects. The combined administration of isoproterenol and the meal had an additive effect on the rise in serum gastrin. Isoproterenol-stimulated gastrin secretion was completely suppressed by propranolol, which had no effect on meal-stimulated gastrin secretion. It is concluded that the mechanism of gastrin hypersecretion in patients with a duodenal ulcer does not involve a specific abnormality of the beta-adrenergic receptor or the receptor that recognizes proteins and their digested products. There is no established role of beta-adrenergic receptor activity in the hypersecretion of gastrin in patients with duodenal ulcers. It is suggested that the beta-adrenergic receptor may have some function unrelated to the acute secretory response of gastrin.

0365 EFFECTS OF PARENTERAL SECRETIN-CHOLECYSTOKININ AND OF DUODENAL ACID PERFUSION ON GASTRIC SECRETION IN DUODENAL ULCER PATIENTS. (Eng.) Butt, J.; Yoshida, T.; Fleshler, B. (800 Stadium Rd., Columbia, MO 65201). *Am. J. Dig. Dis.* 22(4):371-378; 1977.

To investigate the possibility that failure of endogenous hormone(s) release by duodenal acidification might play a role in the genesis of peptic ulcer disease, the effect of parenteral secretin-cholecystokinin and duodenal acid perfusion on broth-stimulated gastric acid secretion was studied in 11 duodenal ulcer patients. Statistically significant inhibition occurred in both experimental conditions. The effect of secretin-cholecystokinin was more marked than the effect of duodenal acid perfusion. A poorly responsive subgroup of patients appeared to be responsible for the diminished inhibitory effect of duodenal acid perfusion. In this poorly responsive group there was a diminished duodenal volume response as well as diminished duodenal acid clearing. It is concluded that there may exist a diminished release of gastrointestinal hormones such as secretin and cholecystokinin in certain duodenal ulcer patients.

0366 DIFFERENCES IN ANTRAL RESPONSE TO MEAT EXTRACT IN DUODENAL ULCER PATIENTS. (Eng.) Perreault, C.; Coutsoftides, T.; Himal, H. S. (Dept. Experimental Surgery, McGill Univ., Montreal, Canada). *Am. J. Gastroenterol.* 67(4):348-353; 1977.

The pathogenesis of duodenal ulcer formation and its relation to acid secretion (basal, antral, and peak) was studied in 59 duodenal ulcer and 12 normal subjects. Antral function was assessed by instilling meat extract solution into the stomach and then measuring acid and serum gastrin output. Peak acid output was significantly higher ($p<0.001$) in the patients (34.14 ± 1.64 mEq/l) than in the normal subjects (20.08 ± 3.79 mEq/l), but basal and peak gastrin values were not significantly different between the two populations. Two groups of duodenal ulcer patients were thus identified: (1) antral responders (31), in whom meat extract caused a significant ($p<0.001$) increase in serum gastrin output (from a basal value of 66.7 ± 7.1 to 153.3 ± 20.5 meat extract-stimulated gastrin output/ml) and acid output (from 4.57 ± 0.54 to 13.33 ± 1.16 mEq/l), and (2) antral nonresponders (28), in whom meat extract did not lead to a significant elevation in gastrin and acid output. The separation of duodenal ulcer patients into those who have or do not have a hyperfunctioning gastric antrum allows one to select the type of operation for each patient. In patients with a hyperfunctioning antrum, vagotomy and antrectomy are suggested; in those with a normal functioning antrum, vagotomy and drainage would be the procedures of choice.

0367 ROUTES OF FEEDING PATIENTS IN THE EARLY POSTOPERATIVE PERIOD IN ULCEROUS DISEASE. (Rus.) Zakharash, M. P. (Kiev Medical Inst., Kiev, USSR). *Vestn. Khir.* 118(5):45-48; 1977.

0368 SIGNIFICANCE OF THE ALIMENTARY FACTOR IN THE GENESIS AND RELAPSE OF PEPTIC ULCER. (Rus.) Budagovskaya, V. N.; Salnikova, G. M. (Inst. Nutrition, Acad. Medical Science, Moscow, USSR). *Vopr. Pitan. (Mosk.)* (3):54-59; 1977.

0369 DYNAMICS OF ACID AND ALKALINE COMPONENTS OF THE GASTRIC CONTENTS DURING THE COURSE OF TREATMENT OF GASTRODUODENITIS AND ULCER DISEASE. (Rus.) Voloshin, A. I.; Troyan, N. F.; Gotsuliak,

O. F. (Chernovtsy Medical Inst., Chernovtsy, USSR). *Vrach. Delo* (6):86-88; 1977.

0370 PATHOPHYSIOLOGY OF PEPTIC ULCER DISEASES. (Fre.) Blum, A. (Stadtspital Triemli, Birmensdorferstrasse 497, 8063 Zurich, Switzerland). *Praxis* 66(18):541-543; 1977.

0371 ALTERATIONS IN GROWTH AFTER GASTRODUODENAL SURGERY: EXPERIMENTAL RESEARCH. (Fre.) Ottolenghi, A.; Perbellini, A.; Montolli, R.; Faccioli, G.; Cappelletto, T.; Bettili, G.; *et al.* (Localita Mure, 37011 Bardolino, Verona, Italy). *Ann. Chir. Infant. Paris* 18(4):299-306; 1977.

0372 TREATMENT OF PEPTIC ULCERATION WITH HISTAMINE H_2-RECEPTOR ANTAGONISTS. (Ger.) Domschke, W.; Domschke, S.; Demling, L. (Med. Univ.-Klinik, Krankenhausstrasse 12, 852 Erlangen, W. Germany). *Fortschr. Med.* 95(31):1899, 1901-1902; 1977.

0373 DISTURBANCES IN THE ORGANISM FOLLOWING GASTRECTOMY. (Pol.) Markert, W. (ul. Zielona 9 m 3, 90-414 Lodz, Poland). *Wiad. Lek.* 30(14):1115-1118; 1977.

0374 ISOENZYMES OF GASTRIC LIPASE IN MAN. (Pol.) Szafran, Z.; Popiela, T.; Wanicka, A.; Szafran, H.; Koscielniak, K. (Acad. Medicine, ul. Wielicka 265, 30-663 Krakow, Poland). *Przegl. Lek.* 34(4):413-417; 1977.

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0376 EPIDEMIOLOGIC INVESTIGATION OF PEPTIC ULCER AMONG COAL MINERS: PART II. EFFECT OF OCCUPATIONAL FACTORS. (Pol.) Zahorski, W.; Marek, K.; Kujawska, A.; Klopotowski, J. (Kliniki Chorob Zawodowych Instytutu Medycyny Pracy, Sosnowcu, Poland). *Med. Prac.* 28(3): 207-216; 1977.

0377 GASTRINEMIA IN NORMAL SUBJECTS AND IN CASES OF DUODENAL ULCER: CORRELATION BETWEEN GASTRIC ACIDITY STIMULATED BY PENTAGASTRIN AND GASTRIN RESPONSE TO TEST MEAL. (Fre.) Fournet, J.; Fouque, B.; Hostein, J.; Maljournal, B.; Bonnet-Eymard, J. (CHU de Grenoble, Pavillon A, 38700 La Tronche, France). *Ann. Gastroenterol. Hepatol.* 13(3):185-194; 1977.

0378 REPEATED OPERATIONS IN PEPTIC ULCER. (Rus.) Trunin, M. A.; Plotnikov, Yu. V. (Leningrad Sanitary-Hygiene Medical Inst., Leningrad, USSR). *Khirurgiia (Mosk.)* (8):3-6; 1977.

0379 FUNCTIONAL CONDITION OF THE GALLBLADDER IN PATIENTS WITH DUODENAL ULCER. (Rus.) Galkin, V. A.; Rubin, M. P.; Khingeeva, S. L. (I. M. Sechenov First Moscow Medical Inst., Moscow, USSR). *Ter. Arkh.* 49(6):81-84; 1977.

0380 PROTEOLYTIC ACTIVITY OF THE STOMACH IN PEPTIC ULCER. (Rus.) Gorshkov, V. A. (Leningrad Sanitary-Hygiene Medical Inst., Leningrad, USSR). *Klin. Med. (Mosk.)* 55(6):53-58; 1977.

0381 FUNCTIONAL CONDITION OF THE GASTRIC MUCOSA IN PATIENTS WITH UNCOMPLICATED PEPTIC ULCER. (Rus.) Kushnir, V. E. (A. A. Bogomoltsa Kiev Medical Inst., Kiev, USSR). *Klin. Med. (Mosk.)* 55(6):49-53; 1977.

0382 TREATMENT OF PATIENTS WITH THE JUVENILE FORM OF PEPTIC ULCER WITH DOCSA PREPARATION. (Rus.) Shirokova, K. I.; Lukasheva, M. V.; Ivanchenkova, R. A.; Smirnova, G. A. (I. M. Sechenov First Moscow Medical Inst., Moscow, USSR). *Klin. Med. (Mosk.)* 55(6):46-49; 1977.

0383 DUODENAL ULCER. I. SELECTION OF EXPLORATORY METHODS. (Fre.) Lambert, R.; Minaire, Y. (Hopital Edouard-Herriot, place d'Arsonval, F 69374 Lyon Cedex 2, France). *Nouv. Presse Med.* 6(7):551-554; 1977.

0384 A NEW APPROACH TO THE STUDY OF PEPTIC ULCER ETIOLOGY BY MEANS OF MULTIDIMENSIONAL STATISTICAL ANALYSIS. (Rus.) Loginov, A. S.; Salnikova, G. M.; Glazkova, T. G. (Central Inst. Gastroenterology, Moscow, USSR). *Ter. Arkh.* 49(2):3-8; 1977.

0385 DIFFERENTIAL DIAGNOSIS OF PEPTIC ULCER, CHRONIC GASTRITIS AND FUNCTIONAL DISEASES OF THE STOMACH IN CHILDREN. (Rus.) Belousov, Iu. V. (Ukrainian Inst. Advanced Medical Studies, USSR). *Pediatriia* (3):31-38; 1977.

0386 POSSIBLE IMPROVEMENT OF DUODENAL BULB ULCER DIAGNOSIS (ACCORDING TO THE DATA ON

LONG-TERM PROPHYLACTIC X-RAY EXAMINATIONS). (Rus.) Labetsky, I. I.; Tager, I. L. (4th Dept. of USSR Ministry of Public Health, Moscow, USSR). *Ter. Arkh.* 49(2):32-36; 1977.

0387 STUDY OF 400 FAMILIES WITH INCREASED INCIDENCE OF GASTRODUODENAL ULCER. DEDUCTIONS CONCERNING THE PATHOGENESIS OF ULCER. (Fre.) Dubarry, J. J. (Hopital Saint-Andre, 1, rue Jean-Burguet, 33000 Bordeaux, France). *Bull. Acad. Natl. Med. Paris* 160(1):54-60; 1976.

See also, 0065, 0097, 0102, 0252, 0292, 0306, 0327, 0335, 0340, 0346, 0349, 0357, 0358, 0361, 0410, 0510, 0737, 0746.

SMALL INTESTINE

0388 THE INTERDIGESTIVE MOTOR COMPLEX OF NORMAL SUBJECTS AND PATIENTS WITH BACTERIAL OVERGROWTH OF THE SMALL INTESTINE. (Eng.) Vantrappen, G.; Janssens, J.; Hellemans, J.; Ghoos, Y. (Dept. Medical Res., Univ. Leuven, Leuven, Belgium). *J. Clin. Invest.* 59(6):1158-1166; 1977.

To investigate whether the interdigestive motor complex identified in several animal species occurs in man, intraluminal pressures were measured in the gastric antrum and at different levels of the upper small intestine in 18 normal subjects. In all normal subjects, the activity front of the interdigestive motor complex was readily identified as an uninterrupted burst of rhythmic contraction waves that progressed down the intestine, and that was followed by a period of quiescence. Quantitative analysis of various parameters of the complex and simultaneous radiological and manometrical observations revealed that it resembled closely the canine interdigestive motor complex. To test the hypothesis that disorders of this motor complex may lead to bacterial overgrowth in the small intestine, similar studies were carried out in 18 patients with a positive $^{14}CO_2$ breath test and in 9 controls with a normal $^{14}CO_2$ breath test. All but five patients had normal interdigestive motor complexes. The five patients in whom the motor complex was absent or greatly disordered had bacterial overgrowth, as evidenced by $^{14}CO_2$ bile acid breath tests before and after antibiotics. These studies establish the presence and define the characteristics of the normal interdigestive motor complex in man. They also suggest that bacterial overgrowth may be due to a specific motility disorder, i.e., complete or almost complete absence of the interdigestive motor complex.

0389 JUXTAPAPILLARY DUODENAL DIVERTICULA AND ABNORMALITIES BY ENDOSCOPIC RETROGRADE CHOLANGIO-PANCREATOGRAPHY (ERCP). (Eng.) Osnes, M.; Myren, J.; Lotveit, T.; Swensen, T. (Ulleval Hosp., Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(3): 347-351; 1977.

The relationship between juxtapapillary duodenal diverticula and changes demonstrated by endoscopic retrograde cholangiopancreatography (ERCP) was investigated. In 174 patients, the biliary and pancreatic ducts were demonstrated by ERCP. Juxtapapillary diverticula were found in 5 of 84 patients with normal duct systems. In 90 cases of ductal abnormalities due to biliary concrements, dilated common bile duct, and pancreatitis, diverticula were found in 35. In patients with abnormalities in both duct systems, diverticula were found in 52%, and in patients with changes in one duct system, diverticula were found in 33%. The difference in occurrence of diverticula in patients with normal duct systems and pathological duct systems was statistically significant ($p<0.02$). The findings indicate a correlation between juxtapapillary diverticula and pathological changes due to biliary concrements, dilated common bile duct without concrements, and pancreatitis.

0390 CURRENT STATUS OF DIAGNOSIS AND MANAGEMENT OF STRANGULATION OBSTRUCTION OF THE SMALL BOWEL. (Eng.) Shatila, A. H.; Chamberlain, B. E.; Webb, W. R. (Southland Medical Building, Suite 128, 6867 Pearl Rd., Cleveland, OH 44130). *Am. J. Surg.* 132(3):299-303; 1976.

Consecutive case records of 50 patients treated for strangulation obstruction of the small bowel are compared with those of 53 patients treated for simple small bowel obstruction. Hernias were the most frequent cause of obstruction in the strangulation group (64%), followed by postoperative adhesions (32%). Postoperative adhesions were the most frequent cause of obstruction in the simple obstruction group (66%) followed by intra-abdominal malignancies (9.0%) and hernias (7.5%). The early differentiation between simple and strangulation obstruction by clinical criteria and laboratory tests was difficult, and strangulation obstruction was suspected preoperatively in only 25 strangulation cases. Abdominal pain, vomiting, abdominal distention, tachycardia, fever, abdominal tenderness, abdominal rebound, and palpable abdominal mass were common to both groups, with incidences of 96, 79, 68, 34, 19, 71, 8, and 9%, resp., in the simple obstruction group and incidences of 90, 86, 60, 44, 24, 86, 10,

and 62%, resp., in the strangulation group. Abdominal wall rigidity (8%), shock (16%), hypothermia (10%), and rectal bleeding (8%) occurred only in the strangulation group and only in patients with late obstruction. Leukocytosis (WBC>11,000/mm^3), 79% neutrophilia or greater, elevated SGOT (>40 μU/ml), elevated LDH (>225 μU/ml), and elevated serum amylase (>200 Somogyi U/ml) were found in 45, 50, 32, 10, and 0%, resp., of the simple obstruction cases and in 62, 70, 46, 37, and 10%, resp., of the strangulation cases. Of 26 patients who had gangrenous bowel preoperatively, the classic signs of strangulation, i.e., fever, leukocytosis, and tachycardia, were absent in 70, 42, and 42%, resp., of these patients. Postoperative mortality was 8% in the strangulation group and 0% in the simple obstruction group. Overall morbidity was 42% in the strangulation group, with wound infections (10 cases), urinary tract infections (8 cases), and pulmonary complications (6 cases) being the most common. Early operation after rapid resuscitation is absolutely indicated for virtually all patients with acute mechanical small bowel obstruction. Patients with obstruction secondary to intra-abdominal carcinomatosis or radiation enteritis and patients with multiple recurrent episodes of intestinal obstruction secondary to adhesions may be excluded and benefit from a more conservative course of therapy.

0391 FAT DIGESTION AFTER JEJUNOILEAL BYPASS OPERATION FOR OBESITY. (Eng.) Sorensen, T. I. A.; Krag, E. (Gentofte Hosp., Univ. Copenhagen, Copenhagen, Denmark). *Scand. J. Gastroenterol.* 11(5):491-495; 1976.

To investigate the etiology of steatorrhea following jejunoileal bypass, fat digestion in the upper jejunum was studied in 12 obese patients treated by jejunoileal bypass. Either 12.5 cm or 37.5 cm of the distal ileum was included in the bypass. The jejunum was intubated, and a test meal was ingested before and 1-2 months after operation. Jejunal aspirates were heated immediately to destroy lipase, pooled, and assayed for dilution marker, bile acids, and total fat. Lipolytic products were determined by thin-layer chromatography and densitometry. The micellar phase was achieved by ultracentrifugation and analyzed for total fat. Both the bile acid concentration and lipolysis decreased after both bypass operations ($p<0.05$ and $p<0.01$, resp.). In accordance with this, micellar solubilization of fat was impaired after the operation ($p<0.01$ after the first type of bypass; $p<0.05$ after the second type.). Bile acid concentration and micellar solubilization were lower after bypass with a long ileal loop than after bypass with a short ileal loop. Fat absorption in the duodenum and upper jejunum did not change significantly after any of the operations. Jejunoileal bypass impairs fat digestion, but this does not contribute significantly to postoperative fat malabsorption.

0392 ACUTE ADHESION-PRODUCED ILEUS. (Pol.) Miszczak, Z. (ul. Grottgera 5, 37-700 Przemysl, Poland). *Wiad. Lek.* 30(13):1001-1004; 1977.

0393 ASSESSMENT OF THE FUNCTIONAL AND MORPHOLOGICAL CONDITION OF THE LIVER IN PATIENTS PREPARED FOR SURGICAL TREATMENT OF OBESITY. (Pol.) Tuszewski, M. (Acad. Medicine, ul. Przybyszewskiego 49, 60-355 Poznan, Poland). *Pol. Tyg. Lek.* 32(36): 1393-1395; 1977.

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0396 HEMOPERITONEUM AS A RARE COMPLICATION OF MECKEL'S DIVERTICULUM: A CASE REPORT. (Ger.) Frank, G.; Reichel, K. (Medizinischen Hochschule Hannover, Karl-Wiechert-Allee 9, D-3000 Hannover 61, W. Germany). *Chirurg* 48(4):238-240; 1977.

0397 A CASE OF LARGE ILEUM CARCINOIDOSIS WITH HEART DISEASE. (Spa.) Lapertosa, G.; Gatteschi, B. (Istituto di Anatomia e Istologia Patologia dell'Universita di Genova, Genova, Italy). *Pathologica* 69(991/992):315-324; 1977.

0398 LATE ILEAL METASTASES FROM A SKIN MELANOMA WITH MAINLY HEMORRHAGIC SYMPTOMS: REPORT OF ONE CASE. (Fre.) Chometowski, S.; Nguyen Cat, R.; Assadourian, R.; Bourde, J.; Lamy, J. (45, rue de Paris, 92220 Bagneux, France). *J. Chir. (Paris)* 113(5/6):537-542; 1977.

0399 DOUBLE-CONTRAST TECHNIQUE FOR DIAGNOSTICS OF THE LARGE INTESTINE. (Ger.) Schilling, T. (Israelitisches Krankenhaus in Hamburg, Orchiedeenstieg 14, 2000 Hamburg 60, W. Germany). *Roentgenblaetter* 30(8):388-399; 1977.

0400 THE BLIND LOOP SYNDROME FOLLOWING INTESTINAL ANASTOMOSIS. (Ger.) Lynen, F. K.; Raguse, T. (St.-Elisabeth Krankenhaus, 5130 Gelsenkirchen, W. Germany). *Aktuel. Chir.* 12(2):247-254; 1977.

0401 THE POSSIBILITIES OF ARTERIOGRAPHY IN ILEOCECAL INVAGINATION. (Cze.) Chvojka,

J. (Rentgenologicke oddeleni Vojenske nemocnice, 304 46 Plzen, Czechoslovakia). *Cesk. Radiol.* 31 (3):172-178; 1977.

0402 RECURRENT MELENA DUE TO INTRA-ILEAL FISSURE OF A FALSE ILIAC ANEURYSM: TREATMENT--CURE. (Fre.) Doutre, L. P.; Masson, B.; Pernot, F.; Houdelette, P. (Clinique Chirurgicale A, 311, boulevard du President Wilson, 33200 Bordeaux, France). *Chirurgie* 103(6):424-429; 1977.

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0404 TECHNICAL FAILURES IN SMALL BOWEL SURGERY. (Ger.) Kummerle, F.; Seitz, W. (Chirurgischen Universitatsklinik Mainz, Langenbeckstr. 1, D-6500 Mainz 1, W. Germany). *Zentralbl. Chir.* 102(17):1045-1053; 1977.

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0406 JEJUNO-ILEAL SHORT-CIRCUIT OPERATIONS IN THE TREATMENT OF OBESITY: 16 PERSONAL CASES. (Fre.) Leger, L.; Moulle, P.; Chiche, B.; Louvel, A. (16, rue de Teheran, 75008 Paris, France). *J. Chir. (Paris)* 113(3):229-248; 1977.

0407 EARLY SURGICAL TREATMENT OF COMPLICATIONS OF JEJUNAL DIVERTICULOSIS. (Fre.) Ansay, J. (Hopital Universitaire Brugmann, 1020 Brussels, Belgium). *Lyon Chir.* 73(3):204-206; 1977.

0408 RADIOLOGICAL EXAMINATION OF SMALL INTESTINE AFTER JEJUNOILEAL BYPASS OPERATION FOR EXTREME OBESITY. (Pol.) Flejsierowicz, Z.; Stawny, B.; Tuszewski, M. (Acad. Medicine, ul. Szkolna 8/12, 61-833 Poznan, Poland). *Pol. Przegl. Med. Nukl.* 41(2):131-134; 1977.

0409 SUTURE TECHNIQUE IN INTESTINAL ANASTOMOSIS. (Ger.) Herzog, B. (Kinderspital, Romergasse 8, CH-4005, Switzerland). *Langenbecks Arch. Chir.* 344(1):1-5; 1977.

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0411 DISEASES OF THE ORGANS OF THE ABDOMINAL CAVITY AND RETROPERITONEAL SPACE SIMULATING ACUTE APPENDICITIS IN CHILDREN. (Rus.) Akzhigitov, G. N. (Volgograd Medical Inst., Volgograd, USSR). *Vestn. Khir.* 119(8):118-121; 1977.

0412 CLINICAL SIGNIFICANCE OF TRANSIT TIME AND REFLUX FOLLOWING SMALL INTESTINAL SHUNT OPERATION. (Ger.) Husemann, B.; Muhrer, K. H.; Holik, B. (Chirurgische Univ.-Klinik, Maximiliansplatz, D-8520 Erlangen, W. Germany). *Chirurg* 48 (1):42-45; 1977.

0413 PEUTZ-JEGHER'S SYNDROME: REPORT OF ONE CASE. (Fre.) Devic, G.; Gelain, J. (46 bld de la Republique, F. 38200 Vienna, Austria). *Lyon Chir.* 73(3):209-210; 1977.

0414 DUODENOGASTRIC REFLUX. (Ger.) Rothmund, M. (Universitat Mainz, Chirurgische Klinik, Langenbeckstr. 1, 6500 Mainz, W. Germany). *Z. Gastroenterol.* 15(3):192-201; 1977.

0415 DECOMPRESSIVE OPERATION IN DUODENAL STASIS. (Rus.) Smirnov, E. V. (No affiliation given). *Khirurgiia (Mosk.)* (3):5-9; 1977.

0416 PERFORATION OF THE SMALL INTESTINE DURING THE PROPHYLAXIS OF THROMBOEMBOLISM WITH OXYPHENOBUTAZONE (TANDERIL). (Fre.) Chaneliere, N.; Bourgeois, M.; Mathieu, M. F. (33 avenue A.-Briand, 39000 Lons-le-Saunier, France). *Lyon Chir.* 73(2):150-151; 1977.

0417 POTASSIUM CHLORIDE AND UNSPECIFIC ULCER OF THE SMALL INTESTINE. (Fre.) Dayer, P.; Cech, P.; Courvoisier, B. (Hopital cantonal, CH-1211 Geneva 4, Switzerland). *Schweiz. Med. Wochenschr.* 107(11):379-380; 1977.

0418 CATAMNESTIC INVESTIGATION OF 82 PATIENTS WITH UNSPECIFIC CHANGES OF THE SMALL INTESTINAL MUCOSA. (Ger.) Morger, H.; Shmerling, D. H. (Kinderspital, Steinwiesstrasse 75, CH-8032 Zurich, Switzerland). *Helv. Paediatr. Acta* 31(6): 453-462; 1976.

0419 INVOLVEMENT OF JOINTS IN INTESTINAL DISEASES. (Ger.) Tanner, E.; Bosseckert, H.; Fritze, Ch.; Seidel, K. (Spezialklinik fur Rheumakranke, DDR-6821 Etzelbach, E. Germany). *Dtsch. Gesundheitsw.* 32(12):529-533; 1977.

0420 CREATION OF INTESTINAL VALVE. A NEW METHOD. (Fre.) Zambouras, D. A. ("Agia Olga" General

State Hosp., N. Ionia-Athens, Greece). *Nouv. Presse Med.* 6(10):847-849; 1977.

0421 TREATMENT OF DIARRHEA SECONDARY TO INTESTINAL RESECTION. (Fre.) Le Quintrec, Y.; Gendre, J. P. (Hopital Rothschild, 43, bd de Picpus, 75571 Paris Cedex 12, France). *Sem. Hop. Ther.* 53(3): 177-180; 1977.

0422 SIMULTANEOUS COLOSTOMY AND ILEOSTOMY: ILEOSTOMY WITH AUTONOMOUS AND CONTINENT SPHINCTER. (Fre.) Palma, E. C. (21 de Setiembre 2385, Montevideo, Uruguay). *Lyon Chir.* 73(4): 311-316; 1977.

See also, 0003, 0085, 0244, 0245, 0247, 0263, 0279, 0283, 0290, 0292, 0297, 0298, 0299, 0300, 0319, 0327, 0338, 0361, 0370, 0386, 0387, 0438, 0451, 0454, 0484, 0695, 0700, 0717, 0722, 0723, 0731, 0732, 0736, 0740, 0756, 0757, 0758.

SMALL INTESTINE

Malabsorption

0423 JEJUNAL PERFUSION OF SIMPLE AND CONJUGATED FOLATES IN CELIAC SPRUE. (Eng.) Halsted, C. H.; Reisenauer, A. M.; Romero, J. J.; Cantor, D. S.; Ruebner, B. (Sch. Medicine, Univ. California, Davis, CA 95616). *J. Clin. Invest.* 59(5):933-940; 1977.

The intestinal absorption of [^{3}H]pteroylmonoglutamate (simple folic acid) and pteroyl-μ[^{14}C]glutamyl-γ-hexaglutamate ([^{14}C]PG-7, conjugated folic acid) was assessed by the method of jejunal perfusion in five patients with proven celiac sprue who were studied after a gluten-containing or a gluten-free diet, and in nine normal subjects. The luminal disappearance of each folate was markedly impaired after exposure of the patients to dietary gluten in comparison with that in patients on a gluten-free diet ($p<0.02$ for [^{3}H]PG-1, $p<0.01$ for [^{14}C]PG-7), but the levels in the celiac patients on the gluten-free diets did not come within the range found in the normal subjects. Column chromatography of the luminal aspirates revealed similar spectra of hydrolytic products of [^{14}C]PG-7, whereas the fraction of the distal aspirate chromatogram appearing as pteroyl-μ[^{14}C]glutamyl-γ-monoglutamate ([^{14}C]-PG-1) was similar in all three groups. By accounting for the variable effects of absorption on the luminal appearance of [^{14}C]PG-1 and by correcting for mucosal hydrolysis that was not followed by release of [^{14}C]PG-1 to the luminal contents, the calculated rate of *in vivo* hydrolysis of [^{14}C]-PG-7 to [^{14}C]PG-1 was found to be impaired in both celiac sprue groups, with significant improvement on treatment. In mucosal biopsies from the sprue patients, the *in vitro* activity of folate conjugase in whole homogenates was higher and the activity of disaccharidase lower than in a group of 12 normal mucosal biopsies. These *in vitro* data suggest that the predominant cellular location of mucosal folate conjugase is different from that of disaccharidase, whereas comparison with the results of *in vivo* hydrolysis suggests that measurement of the enzyme in whole mucosal homogenates overestimates its significant digestive activity. These studies indicate that the mucosal lesion of celiac sprue significantly limits the intestinal absorption of both simple and conjugated folate, and that malabsorption of conjugated folate results from a combination of impaired hydrolysis and decreased mucosal uptake of hydrolytic product.

0424 PHARMACOKINETICS OF SALICYLATE AND INDOMETHACIN IN COELIAC DISEASE. (Eng.) Parsons, R. L.; Kaye, C. M.; Raymond, K. (Guy's Hosp., St. Thomas St., London SE1 9RT, England). *Eur. J. Clin. Pharmacol.* 11(6):473-477; 1977.

To determine whether the abnormal plasma concentration/time curve of lipid soluble drugs seen in celiac disease is related to their pKa, the plasma concentrations of salicylate and indomethacin were measured after a single p.o. dose of aspirin (600 mg) or indomethacin (50 mg) in 12 starved normal subjects and in 12 adult patients with celiac disease. The absorption of salicylate in the celiac patients was faster than that in the normal subjects. The plasma concentration/time curve of indomethacin in both groups was similar during the absorption phase, but there were significant differences between the groups in its elimination. The abnormal absorption pattern of salicylate in celiac disease does not appear to be related to its pKa. Possible causes of the difference in salicylate absorption include changes in gastric emptying or altered small intestinal permeability.

0425 DEPRESSED CELL-MEDIATED IMMUNITY IN COELIAC DISEASE. (Eng.) Scott, B. B.; Losowsky, M. S. (St. James' Hosp., Leeds, England). *Gut* 17(11): 900-905; 1976.

Phytohemagglutinin (PHA)-induced lymphocyte trans-

formation was used to assess the integrity of cell-mediated immunity (CMI) in 10 untreated celiac patients on a normal diet, 14 celiac patients treated with a gluten-free diet (GFD), and 13 healthy controls. Untreated celiac patients had significantly reduced transformation compared with controls in both autologous and pooled AB serum ($p<0.02$). Treated celiac patients had significantly reduced transformation in autologous serum ($p<0.05$) but not in pooled AB serum ($p>0.1$). Responses to PHA in treated patients were significantly higher than in untreated patients in pooled AB serum ($p=0.03$) but not in autologous serum ($p=0.06$). Responses to PHA in pooled AB serum were significantly higher than in autologous serum in patients on a GFD ($p=0.04$) but not in celiac patients on a normal diet ($p=0.16$). The improvement in CMI after treatment was particularly apparent in six celiac patients studied on both a normal diet and a GFD. Lymphocytotoxic antibodies were detected in only three patients, and lymphocyte transformation was more depressed in autologous serum in two of these patients. No correlation was established between lymphocyte transformation and nutritional deficiency. Only 22% of the celiac patients had positive Mantoux tests compared with 85% of the controls ($p<0.001$). Two patients who were tested before and after *Bacillus Calmette-Guerin* vaccination remained negative, although one had responded well to the GFD. Mantoux tests were as commonly negative in treated as in untreated patients. CMI depression in celiac disease may be partly explained by an undefined depressing serum factor. If depressed immunity in celiac disease is related to the development of malignancy, the case for a strict GFD in all patients is reinforced by the improved CMI as assessed by lymphocyte transformation in GFD-treated patients.

0426 CELL-MEDIATED AUTOIMMUNITY IN COELIAC DISEASE. (Eng.) Scott, B. B.; Losowsky, M. S. (Univ. Dept. Medicine, St. James' Hosp., Leeds, LS9 7TF, England). *Clin. Exp. Immunol.* 26(2):243-246; 1976.

Lymphocyte transformation in response to a crude freeze-dried preparation of whole jejunal mucosa was studied in 15 patients with celiac disease and compared with the findings in 15 age-matched controls. The transformation ratio was markedly increased in three patients. The patients with the highest and third highest ratios were on a gluten-free diet, while the other responders were on a normal diet. These observations suggest the possible existence of cell-mediated autoimmunity to jejunal mucosa in some patients with celiac disease and might be relevant to the development of some of the complications of the disease.

0427 HYPOTHYROIDISM AND COELIAC DISEASE. A FAMILY STUDY. (Eng.) McCarthy, D.; Manning, N.; Rees, J. P. R.; Drury, M. I. (Mater Misericordiae Hosp., Dublin, Ireland). *Ir. J. Med. Sci.* 145(7):237-238; 1976.

0428 VITAMIN B_6 NUTRITURE OF CHILDREN WITH ACUTE CELIAC DISEASE, CELIAC DISEASE IN REMISSION, AND OF CHILDREN WITH NORMAL DUODENAL MUCOSA. (Eng.) Reinken, L.; Zieglauer, H.; Berger, H. (Dept. Pediatrics, Univ. Innsbruck, Innsbruck, Austria). *Am. J. Clin. Nutr.* 29(7):750-753; 1976.

0429 CEROID ENTEROPATHY AND VITAMIN E DEFICIENCY. (Eng.) Lee, S. P.; Nicholson, G. I. (Auckland Hosp., Auckland, New Zealand). *N.Z. Med. J.* 83(563):318-320; 1976.

0430 POLYMYOSITIS AND OTHER MYOPATHIES AS PRINCIPAL FEATURE IN GLUTEN-SENSITIVE ENTEROPATHY [Abstract]. (Eng.) Hallert, C.; Walan, A.; Henriksson, K. G. (Dept. Internal Medicine, Univ. Linkoping, S-581 85 Linkoping, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):30; 1977.

0431 BILE SALT ABSORPTION IN COELIAC DISEASE [Abstract]. (Eng.) Gillberg, R.; Andersson, H. (Sahlgren's Hosp., Goteborg, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):24; 1977.

0432 THE NATURE OF GLUTEN TOXICITY IN COELIAC DISEASE [Abstract]. (Eng.) Fottrell, P. F.; Phelan, J.; Stevens, F.; McNicholl, B.; McCarthy, C. F. (Dept. Biochemistry, Univ. Coll., Galway, Ireland). *J. Anat.* 124(2):490: 1977.

0433 EFFECT OF FOLATE DEFICIENCY ON MUCOSAL DNA SYNTHESIS DURING RECOVERY FROM TROPICAL MALABSORPTION (TM) [Abstract]. (Eng.) Tomkins, A. M.; McNurlan, M.; Wright, S. (Hosp. Tropical Diseases, London, England). *Gut* 18(11): A945; 1977.

0434 INCREASED PREVALENCE OF EPILEPSY IN COELIAC DISEASE [Abstract]. (Eng.) Laidlow, J. M.; Chapman, R. G.; Colin-Jones, D. G.; Eade, O.; Smith, C. L. (Royal Hosp., Portsmouth, England). *Gut* 18(11):A943; 1977.

0435 CHOLECYSTOKININ ABNORMALITIES IN COELIAC DISEASE [Abstract]. (Eng.) Polak, J. M.; McCrossan, M. V.; Timson, C. M.; Johnston, D. R.; Hudson, D.; Szelke, M.; *et al.* (Royal Postgraduate Medical Sch., London, England). *Gut* 18(11):A942; 1977.

0436 SERIAL LIGHT AND ELECTRON MICROSCOPY OF INTESTINAL MUCOSA IN WHIPPLE'S DISEASE [Abstract]. (Eng.) Mills, P.; More, I.; Dick, A.; Holden, R.; Watkinson, G. (Western Infirmary, Glasgow, Scotland). *Gut* 18(11):A970; 1977.

0437 DIAGNOSIS OF HYPERSENSITIVITY TO COW'S MILK IN DIGESTIVE TRACT DISEASES. (Pol.)

Waligora, J.; Martynow, S.; Fedorenko, T.; Fedorenko, W. (Instytutu Medycyny we Lwowie, Lvov, USSR). *Pol. Przegl. Chir.* 49(6):599-603; 1977.

0438 GALLBLADDER INERTIA AND CELIAC DISEASE: EFFECT OF GLUTEN-FREE DIET ON GALLBLADDER FUNCTION. (Fre.) Delamarre, J.; Joly, J. P.; Capron, J. P.; Chivrac, D.; Dupas, J. L.; Gontier, M. F.; Lorriaux, A. (Centre Hospitalier Universitaire, place Victor-Pauchet, F 80030 Amiens, France). *Gastroenterol. Clin. Biol.* 1(4):365-368; 1977.

See also, 0016, 0125, 0288, 0301, 0388, 0391, 0403, 0406, 0419, 0504, 0675, 0718, 0740, 0755, 0756.

LARGE INTESTINE

0439 GEOGRAPHIC PATTERNS OF LARGE BOWEL CANCER IN THE UNITED STATES. (Eng.) Blot, W. J.; Fraumeni, J. F.; Stone, B. J.; McKay, F. W. (Natl. Cancer Inst., U.S. Dept. Health, Education, and Welfare, Bethesda, MD 20014). *J. Natl. Cancer Inst.* 57(6):1225-1231; 1976.

Age-adjusted colon and rectal cancer mortality rates during 1950-1969 were correlated by sex and race (white and nonwhite) with demographic data for 3,056 counties of the contiguous United States. Mortality from both types of cancer was consistently elevated in counties with large populations and with higher income and education levels. Geographically, mortality rates for both types of cancer were highest in the northeast and lowest in the southeast. The death rates among whites with colon cancer were generally higher for women than for men, except in highly populous counties where the situation was reversed. Colon cancer mortalities were highest in counties with relatively high percentages of people of Irish, Czechoslovak, or German descent. The death rate for rectal cancer among whites was greater among men than among women. However, the male/female mortality ratio for rectal cancer rose with increasing population size, similar to a pattern seen for colon cancer. Rectal cancer death rates among whites were highest in counties with a high percentage of residents of Greek, Irish, and German stock. Colorectal cancer mortality rate patterns for nonwhites of both sexes resembled those for whites. Colon cancer mortality rates in both sexes were higher among whites than nonwhites, especially in the north. Rectal cancer mortality rates in men likewise were higher among whites than nonwhites in the north; however, no racial differences were found in the south. For women, the rectal cancer death rates were slightly higher among whites in the north but slightly higher among nonwhites in the south. Colon cancer mortality among nonwhites was higher in women than in men, except in populous northern counties where the rates were about equal. Rectal cancer was more common among men than among women in nonwhites, though the sex difference was not as great as it was among whites. A survey on dietary habits by region and on alcohol sales by county failed to show an impressive correlation with bowel cancer mortality. The geographical variations in colorectal cancer mortality rate may be helpful in the identification of dietary and other environmental determinants of large bowel tumors.

0440 LINCOMYCIN-CLINDAMYCIN COLITIS IS NOT AN ENTITY. (Eng.) Devroede, G.; Poisson, J.; Madarnas, P.; Beaudry, R.; Haddad, H.; Strom, B.; *et al.* (Centre Hospitalier Universitaire Sherbrooke, Quebec J1H 5N4, Canada). *Can. J. Surg.* 20(4):326-335; 1977.

From 1969 to 1975, 10 cases of pseudomembranous colitis (PC, group I) and 13 cases of nonpseudomembranous colitis (NPC, group II) developed as a result of ingestion of antibiotics. All patients recovered after the administration of clindamycin plus steroids. During this period, an erroneous diagnosis of PC was made in three patients (later diagnosed as having ulcers) who had not received antibiotics but had focal accumulation of pus on the rectal mucosa and were chronically ill. The penicillin class of antibiotics was responsible for colitis in six group I patients and five in group II, compared with three and five patients, resp., for the lincomycin-clindamycin class. Patients with PC were more seriously ill than those with NPC, and their disease lasted longer (31 versus 15 days, $p<0.01$). PC patients had more frequent abdominal distention ($p<0.001$), a faster pulse rate ($p<0.05$), and a higher temperature ($p<0.001$) or one of longer duration ($p<0.001$). The WBC count was higher in PC patients ($p<0.005$), and values for serum protein ($p<0.001$), hemoglobin ($p<0.005$), potassium ($p<0.001$), sodium ($p<0.05$), and total carbon dioxide ($p<0.025$) were lower. Seven NPC patients had target lesions in the rectosigmoid mucosa. At follow-up endoscopy, pseudomembranes were not present even if the disease had not been of shorter duration ($p>0.05$) than that of the PC patients. Although NPC and PC have different clinico-pathological manifestations, they may be of identical etiology. It is concluded that use of the term lincomycin-clindamycin colitis is unjustified.

0441 INTRALUMINAL PRESSURE IN THE SIGMOID COLON. II. PATIENTS WITH SIGMOID DIVER-

TICULA AND RELATED CONDITIONS. (Eng.) Weinreich, J.; Andersen, D. (Surgical Univ. Clinic I, Aarhus Amtssygehus, DK-8000 Aarhus C, Denmark). *Scand. J. Gastroenterol.* 11(6):581-586; 1976.

Sigmoid pressure activity was measured in 188 patients with various colonic diseases to determine whether pathophysiological similarities exist in these entities. The clinical syndromes included chronic diverticular disease (62 patients), colonic diverticulosis (15), atypical diverticular disease (36), acute diverticular disease (26), colicky sigmoid syndrome (15), sigmoid syndrome with predominant diarrhea (8), sigmoid syndrome with predominant constipation (16), and sigmoid syndrome with vague dyspepsia (10). The last four groups refer to what is generally known as the irritable bowel syndrome. Intraluminal sigmoid pressure was determined after the administration of neostigmine (0.5 mg, i.v.). Analysis of variance indicated that patients with chronic diverticular disease, patients with acute diverticular disease, and patients with the colicky sigmoid syndrome all might belong to a single population with respect to pressure activity; the median motility indices in the three groups were 1,996, 1,469, and 1,815, resp. Patients in the other categories showed pressure activities similar to those previously reported in normal subjects.

0442 TOTAL BILE ACIDS IN PORTAL VENOUS SERUM: A QUALITATIVE AND QUANTITATIVE ASSESSMENT [Abstract]. (Eng.) Holzbach, R. T.; Marsh, M. (Cleveland Clinic Foundation, Cleveland, OH). *Gastroenterology* 73(5):1225; 1977.

0443 MALLORY-WEISS LESION FOLLOWING CANCER CHEMOTHERAPY [Letter to Editor]. (Eng.) Enck, R. E. (William Beaumont Army Medical Center, El Paso, TX 79920). *Lancet* 2(8044):927-928; 1977.

0444 ERYTHEMA NODOSUM PRESENTING BEFORE INFLAMMATORY BOWEL DISEASE [Letter to Editor]. (Eng.) Linaker, B. D. (Broadgreen Hosp., Liverpool, England). *Lancet* 2(8044):931; 1977.

0445 INSTRUMENTAL-ENDOSCOPIC-PERFORATIONS OF THE COLON AND RECTUM [Abstract]. (Eng.) Filler, D.; Hermstein, N.; Groitl, H. (Dept. Surgery, Univ. Giessen, Giessen, W. Germany). *Endoscopy* 9(3):195; 1977.

0446 EXPERIENCES IN 528 COLOSCOPIC POLYPECTOMIES [Abstract]. (Eng.) Otto, P.; Bunnemann, H.; Huchzermeyer, H.; Muller, H. (Kreiskrankenhaus Grossburgwedel, Hannover, W. Germany). *Endoscopy* 9(3):194; 1977.

0447 COLONOSCOPE TYPE CF-LB3 (OLYMPUS)--A ROUTINE INSTRUMENT FOR COMPLETE COLOSCOPY [Abstract]. (Eng.) Mockel, W. (Innere Abt. Ev. Krankenhaus Koln-Kalk, Koln, W. Germany). *Endoscopy* 9(3):193; 1977.

0448 PROCTOSIGMOIDOSCOPY IN CHILDREN. ANALYSIS OF RESULTS [Abstract]. (Eng.) da Silva Kotze, L. M. (No affiliation given). *Arq. Gastroenterol.* 14(3):159; 1977.

0449 EXPLOSION OF HYDROGEN GAS IN THE COLON DURING PROCTOSIGMOIDOSCOPY. (Eng.) Bond, J. H.; Levy, M.; Levitt, M. D. (Minneapolis Veterans Admin. Hosp., Minneapolis, MN). *Gastrointest. Endosc.* 23(1):41-42; 1976.

0450 FUNCTION STATE OF INTESTINE IN COLONIC POLYPOSIS (A CLINIC RADIOLOGICAL EXAMINATION. (Ger.) Keves, E. L.; Pyteva, G. P.; Bychovskaja, A. M.; Katorkin, E. N. (Kujbyschewer Med. D.I. Uljanow Inst., Kujbyschev, USSR). *Radiol. Diagn. (Berl.)* 17(3):401-407; 1976.

0451 CLINICAL FEATURES OF NEONATAL OCCLUSION: MANAGEMENT. (Fre.) Frappat, P.; Dyon, J. F.; Baudain, Ph. (No affiliation given). *Rev. Fr. Gastroenterol.* (118):7-51; 1976.

0452 IMPORTANCE OF CHANGES IN THE GASTROINTESTINAL MUCOSA IN DISTURBANCES OF THE MEDIATOR MECHANISM IN ALLERGIC AND DYSENTERY COLITIS. (Rus.) Shenkman, B. Z. (Saratov Medical Inst., Saratov, USSR). *Patol. Fisiol. Eksp. Ter.* (3):77-79; 1977.

0453 INTESTINAL INTUBATION THROUGH 9 GASTROSTOMA IN ILEUS IN CHILDREN. (Rus.) Gadzhimirzaev, G. A.; Arbuliev, M. G.; Ashurbekov, V. T.; Medzhidov, R. (Dagestan Medical Inst., Makhachkala, USSR). *Khirurgiia (Mosk.)* (8):42-45; 1977.

0454 TREATMENT OF PATIENTS WITH EXTERNAL INTESTINAL FISTULAS. (Rus.) Maloman, E. N.; Zhabanovskii, V. E. (Kishinev Medical Inst., Kishinev, USSR). *Khirurgiia (Mosk.)* (8):45-50; 1977.

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CITIS IN YOUNG PERSONS. (Rus.) Podonenko-Bogdanova, A. P. (Donetsk Medical Inst., Donetsk, USSR). *Vestn. Khir.* (6):70-72; 1977.

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0461 RETROPERITONEAL SUPPURATION SECONDARY TO PERFORATION OF AN ISOLATED DIVERTICULUM OF THE CECUM. (Fre.) Lagache, G.; Combemale, B.; Richet, F.; Bahon le Capon, J. (No affiliation given). *Chirurgie* 103(5):301-304; 1977.

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Barret, A.; Vaysse, C.; Mason, J.; Gedeon, A. (Clinique Chirurgicale C, Toulouse, France). *Chirurgie* 103(6):430-439; 1977.

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0479 ANALYSIS OF 175 OPERATIONS FOR HIRSCH-SPRUNG'S DISEASE IN CHILDREN. (Rus.) Sitkovsky, N. B.; Baklanova, A. N. (Kiev Medical Inst., Kiev, USSR). *Klin. Khir.* (6):6-9; 1977.

0480 GENERALIZED FECAL PERITONITIS: ETIOPATHOGENIC AND THERAPEUTIC CONSIDERATIONS IN 30 CASES. (Fre.) Hollender, L. F.; Meyer, Ch.; Calderoli, H.; Zavaletta, D. (Service de Chirurgie Generale 3, C.H.U., 1, place de l'Hopital, 67005 Strasbourg Cedex, France). *J. Chir. (Paris)* 113(4): 327-336; 1977.

0481 FAMILIAL COLONIC POLYPOSIS. (Fre.) Granges, Y.; Montant, A.; Megevand, R. (Clinique Sainte-Claire, 3960 Sierre, Switzerland). *J. Chir. (Paris)* 113(4):395-404; 1977.

0482 PROBLEMS RAISED BY THE PREVENTION AND DETECTION OF COLONIC AND RECTAL CARCINOMA. (Fre.) Frexinos, J.; Escourrou, J.; Joffre, F.; Suduca, P.; Ribert, A. (Centre Hospitalier Universitaire de Rangueil, 31054 Toulouse Cedex, France). *Sem. Hop. Paris* 53(24):1462-1468; 1977.

0483 CECOSTOMY AND LEFT COLECTOMY. (Fre.) Pouyet, M.; Perrin, J. P.; Treboux, M. (Clinique Claude Bernard, 69600 Ouillins, France). *Lyon Chir.* 73(3):206-208; 1977.

0484 INVASION OF THE DUODENAL WALL BY CARCINOMA OF THE RIGHT COLON. (Fre.) Guillemin, G.; Braillon, G.; Rivier, R.; Bigay, D. (Clinique Chirurgicale C, Hotel-Dieu, 69288 Lyon Cedex 2, France). *Lyon Chir.* 73(2):106-110; 1977.

0485 PROFUSE COLONIC HEMORRHAGE DUE TO DIVERTICULOSIS: ROLE OF ARTERIOGRAPHY. (Fre.) Marre, Ph.; Paillot, J.; de Hochepied, F.; Alexandre, J. H. (Hopital Broussais, 96, rue Didot, 75014 Paris, France). *Ann. Chir.* 31(6):505-510; 1977.

0486 A RARE CASE OF ACUTE BLEEDING FROM A STRESS COLON: ETIOLOGY, PATHOGENESIS, AND TREATMENT. (Fre.) Mercati, U.; Vecchiarelli, G.; Berardi, F.; Natalini, G. (Hopital Regional, Perouse, Ombrie, Italy). *J. Chir. (Paris)* 113 (5/6):531-536; 1977.

See also, 0010, 0012, 0237, 0245, 0283, 0299, 0300, 0304, 0305, 0327, 0350, 0398, 0399, 0400, 0401, 0411, 0421, 0422, 0490, 0491, 0492, 0717, 0722, 0723, 0731, 0732, 0733, 0736, 0756, 0761.

LARGE INTESTINE

Ulcerative Colitis

0487 BLOOD pH: A TEST FOR ASSESSMENT OF SEVERITY IN PROCTOCOLITIS. (Eng.) Caprilla, R.; Vernia, P.; Colanieri, O.; Torsoli, A. (Policlinico Umberto I, Rome, Italy). *Gut* 17(10):763-769; 1976.

Clinical and laboratory studies on 58 patients with active idiopathic proctocolitis (complicated by toxic megacolon in 10 cases) are reported. As the severity and extent of disease increased, arterial pH became progressively higher. In cases of mild and moderate disease, mean pH values of about 7.43 and 7.42, resp., were observed; a pH over 7.50 invariably indicated severe colitis or toxic megacolon and usually corresponded with total colitis. Various degrees of correlation were found between pH and pulse rate (r=0.6036), body temperature (r= 0.4806), ESR (r=0.4826), and plasma albumin (r= 0.5778). In cases of mild, moderate, severe, and complicated (toxic megacolon) disease, the maximum body temperatures were 36.6 ± 0.07, 36.8 ± 0.16, 37.3 ± 0.15, and 37.6 ± 0.35 C, resp. The maximum pulse rates for the above disease categories were 78.8 ± 2.05, 82.7 ± 2.37, 93.8 ± 3.46, and 100.0 ± 6.6/min, resp. The ESR values (1st hr) for these disease categories were 9.08 ± 1.9, 30.3 ± 9.4, 27.7 ± 6.1, and 44.7 ± 9.0 mm, resp. Serum albumin levels for the disease categories were 3.75 ± 0.11, 3.23 ± 0.10, 2.57 ± 0.15, and 2.38 ± 0.22 g/100 ml, resp. A correlation was also found between arterial

pH and gas in the small and large bowel (r=0.5652). Intestinal gas levels in mild, moderate, severe, and complicated disease were 54.4 ± 4.81, 75.1 ± 6.93, 139.3 ± 18.5, and 339.3 ± 27.8 cm^2, resp. Blood pH measurement and plain x-ray films of the abdomen are the most reliable single methods for rapid detection of the early phases of intestinal dilatation. A progressively increasing amount of intestinal gas and a blood pH above 7.50 should be regarded as alarm signs that toxic dilatation of the colon is impending.

0488 RISK FACTORS IN ULCERATIVE COLITIS. (Eng.) Gilat, T.; Lilos, P.; Benaroya, P.; Zemishlany, Z.; Ribak, J. (Tel Aviv Univ. Sackler Sch. Medicine, Tel Aviv, Israel). *Digestion* 14(5/6):400-408; 1976.

Risk factors were analyzed in 504 Jewish patients with ulcerative colitis (UC). The incidences of severe attacks, weight loss, iron deficiency, liver disease, and arthritis were significantly ($p<0.05$) higher in women (27.0, 33.3, 32.4, 4.9, and 7.4%, resp.) than in men (16.5, 27.3, 25.4, 1.5, 2.3%, resp.). The severity of several parameters of UC tended to be in the following order by community group: Ashkenazi>Sephardi>Oriental. Mortality ($p<0.001$), weight loss ($p=0.05$), and duodenal ulcer ($p=0.01$) were significantly higher or more frequent in patients whose disease started after age 50 yr. Severe attacks were recorded in 25.1% of cases with definite UC and in 5.5% with probable UC ($p<0.001$). In definite UC, the extent of colon affected was distal in 31.6%, substantial in 41.1%, and universal in 16.2% (uncertain in 11.1%) of the cases; the corresponding incidences of extent in probable UC were 53.1, 22.4, and 2.2% (uncertain, 22.3%), resp. ($p< 0.001$). Toxic megacolon and perforation occurred more frequently with increasing disease extent, but the small numbers involved were unsuitable for statistical analysis. Colectomy and mortality were significantly ($p<0.001$) more frequent with increasing disease extent; the incidence of colectomy in universal disease cases was 20.3% versus 6.2% for all cases, and the mortality incidence in universal disease was 24.6% versus 10.7% for all cases. Incidence of severe attacks was positively related to disease extent ($p<0.001$), with the incidences for uncertain disease extent and all cases being 16.7% and 21.6%, resp. The course of the disease was correlated positively with extent of colon affected ($p<0.001$). Other positive correlations found were length of follow-up and disease extent ($p<0.001$) and length of follow-up and disease severity ($p<0.05$).

0489 ULCERATIVE COLITIS IN CHILDREN: RESULTS OF COLECTOMY IN 38 PATIENTS WITH CHILDHOOD ONSET OF DISEASE. (Eng.) Naas, R.; Gruner, O. P. N.; Flatmark, A.; Fretheim, B.; Gjone, E. (Rikshospitalet, Oslo, Norway). *Z. Kinderchir.* 21(3):237-244; 1977.

Ulcerative colitis was studied in children (38 colectomized patients) using ileostomy (14 patients) and ileorectal anastomosis (IRA, 24 patients). A majority of the children were growth retarded at the time of operation. Only 6 of the 38 children had fulminating disease. Duration of the disease before operation was an average of 5.7 yr. The follow-up period (range 18 months-14 yr) ended at the age of 21 yr. Colectomy as late as 16 yr of age had a positive effect on stature ($p<0.01$). Cancer occurred in four IRA patients and appears to be a much greater risk in patients with childhood onset of the disease. Only 6 of the 20 survivors obtained a satisfactory bowel function. Relaparotomy was necessary in 13 of the IRA patients but in none of the ileostomy patients. Ileostomy is a safer operation than IRA and has a significantly lower frequency of complications ($p=0.005$) leading to relaparotomy. Hence, 17 of the 24 IRA patients were either dead, converted to ileostomy, or had an unsatisfactory functional result. Ileostomy function, measured as the interval between changing of the bag, was 1-7 days. Ileostomy function was better ($p=0.0004$) in young patients than in patients after puberty. All survivors except one were able to work, and six of them married. Young patients with ileostomy adapted well and were reported to have fewer problems in their daily life than patients with later onset of disease. Therefore, young age is not a contraindiaction for proctocolectomy and ileostomy.

0490 GLUCOSAMINE SYNTHETASE ACTIVITY OF THE COLONIC MUCOSA IN ULCERATIVE COLITIS AND CROHN'S DISEASE. (Eng.) Goodman, M. J.; Kent, P. W.; Truelove, S. C. (Dept. Medicine, Univ. Chicago, IL). *Gut* 18(3):219-228; 1977.

A miniaturizing method for assay of glucosamine synthetase (GS), requiring just over 4 mg of tissue obtained by rectal biopsies, is described. The study was undertaken to identify differences in GS levels between ulcerative colitis (UC) and Crohn's disease (CD). The mean levels of GS in normal human colonic mucosa (25 colectomy specimens and 20 rectal biopsies) were 13.77 ± 3.97 µmol glucosamine synthesized/hr/g wet weight. There was a lower mean of GS levels in the colectomy specimens from patients with UC and CD, which was related to diminution in the epithelial cell density. However, in samples of mucosa in which the epithelial cells were absent, the GS levels were 3.0 ± 0.9 µmol/hr/g. GS levels were normal when there was loss of goblet cells only. In 15 of the 20 patients recovering from an acute attack of UC, the GS level rose to a peak above the normal range (mean peak level 23.94 µmol/hr/g) followed by a fall to within normal range. This effect did not occur in five patients who did not make prompt recovery. The GS levels in 13 biopsies from patients with CD sparing rectum had a mean of 20.55 µmol/hr/g, considerably higher than that of the normal range ($p< 0.001$). The peak effect in UC appears to be a manifestation of recovery and probably is related to the synthesis of gastrointestinal mucus or immunoglobulin or the secretory component of IgA, all of which contain large amounts of *N*-acetyl-glucosamine.

0491 THE RADIOLOGICAL INVESTIGATION OF COLONIC EPITHELIAL DYSPLASIA AND LATENT CARCINOMA IN CHRONIC ULCERATIVE COLITIS--PRELIMINARY OBSERVATIONS [Abstract]. (Eng.) Frank, P. H.; Riddell, R. H.; Levin, B.; Feczko, P. J. (Dept. Radiology, Univ. Chicago, Chicago, IL). *Clin. Res.* 25(4): 607A; 1977.

0492 SURVEY OF PROTEIN NUTRITION IN INFLAMMATORY BOWEL DISEASE--A RATIONAL BASIS FOR NUTRITIONAL THERAPY [Abstract]. (Eng.) Pickford, I.; Hill, G. L.; Blackett, R. L.; Bradley, J. A. (General Infirmary, Leeds, England). *Gut* 18(11):A970; 1977.

0493 DIRECT METHOD OF MEASURING FAECAL PROTEIN LOSS IN PATIENTS WITH ULCERATIVE COLITIS [Abstract]. (Eng.) Buckell, N. A.; Gould, S. R.; Hernandez, M. A.; Kohn, J.; Lennard-Jones, J. E.; Powell-Tuck, J.; *et al.* (St. Mark's Hosp., London, England). *Gut* 18(11):A971; 1977.

0494 FURTHER STUDIES OF SALICYLAZOSULPHAPYRIDINE METABOLISM IN THE TREATMENT OF ULCERATIVE COLITIS [Abstract]. (Eng.) Cowan, G. O.; Das, K. M.; Eastwood, M. A. (Western General Hosp., Edinburgh, Scotland). *Gut* 18(11):A972; 1977.

0495 CONTROLLED COMPARISON OF SODIUM CROMOGLYCATE AND SULPHASALAZINE IN THE MAINTENANCE OF REMISSION IN ULCERATIVE COLITIS [Abstract]. (Eng.) Dronfield, M. W.; Langman, M. J. S. (City Hosp., Nottingham, England). *Gut* 18(11):A973; 1977.

0496 ENDOCARDITIS COMPLICATING ULCERATIVE COLITIS [Letter to Editor]. (Eng.) Ward, R. L. (Royal Infirmary, Blackburn, Lancaster BB2 3LR, England). *Gastroenterology* 73(5):1189-1190; 1977.

0497 HISTOPATHOLOGICAL OBSERVATIONS IN RECTAL BIOPSIES OF CHILDREN WITH ULCERATIVE COLITIS [Abstract]. (Eng.) Guastavino, E.; Costa, J. A.; Ortiz, J.; Toccalino, H. (No affiliation given). *Arq. Gastroenterol.* 14(3):159; 1977.

0498 COLOSCOPY--ITS VALUE IN DIAGNOSING ULCERATIVE COLITIS AND CROHN'S DISEASE [Abstract]. (Eng.) Weingart, J.; Fischer, L.; Kahl, D.; Ottenjann, R. (Stadt. Krankenhauses Munchen-Neuperlach, Munchen, W. Germany). *Endoscopy* 9(3):194; 1977.

0499 THERAPY OF ULCERATIVE COLITIS. (Ita.) Palmas, S. (Primario II Divisione di Medicina, OO.RR. Sassari, Italy). *Clin. Ter.* 81(5):463-479; 1977.

0500 THE NATURE OF ENDOALLERGY IN CHILDREN WITH NONSPECIFIC ULCERATIVE COLITIS. (Rus.) Kanshina, O. A.; Artemkin, V. D.; Kanshin, N. N. (Moscow Scientific Res. Inst. Epidemiology and Microbiology, Moscow, USSR). *Sov. Med.* (5):36-40; 1977.

0501 EFFICIENCY OF INTRAPORTAL TRANSFUSION THERAPY OF PATIENTS WITH SEVERE UNSPECIFIC ULCERATIVE COLITIS. (Rus.) Novikova, R. I.; Zavgorodny, L. G.; Pedenko, V. P. (Donetsk Medical Inst., Donetsk, USSR). *Klin. Khir.* (8):82-84; 1977.

0502 THE TREATMENT OF ACUTE ATTACKS OF ULCERATIVE COLITIS. (Spa.) Herrerias, J. M.; Perez Ruilopez, M. A.; Pellicer Bautista, F. J.; Garrido Peralta, M. (Hospital Universitario de Sevilla, Seville, Spain). *Rev. Esp. Enferm. Apar. Dig.* 50(5):541-548; 1977.

0503 ULCERATIVE COLITIS AND PREGNANCY. (Cze.) Bitter, J. (I. interni oddeleni krajske nemocnice, 400 01 Usti nad labem, Czechoslovakia). *Cas. Lek. Cesk.* 115(37):1133-1135; 1976.

See also, 0234, 0285, 0419, 0444, 0459, 0711, 0760, 0767, 0773.

PANCREAS

0504 CHYMOTRYPSIN OUTPUT IN THE STOOLS IN PANCREATIC AND OTHER DISEASES. (Eng.) Gilat, T.; Gelman-Malachi, E.; Ronen, O. (Ichilov Hosp., Weizman St., Tel Aviv, Israel). *Am. J. Gastroenterol.* 66(2):140-145; 1976.

The value of a method in which chymotrypsin output in the stools is calculated from analysis of single stool samples, using cuprous isothiocyanate as a continuous marker, was assessed in 16 patients with and 45 patients without pancreatic disease. The calculated values agreed favorably with those from 3-day stool collections. In 45 controls, with and without steatorrhea, the chymotrypsin output was above 10,000 U/24 hr. In six patients with pancreatic insufficiency, the values were considerably lower; all were below 4,000 U/24 hr. In 10 patients with pancreatic disease without insufficiency, the

values were in the normal range (>10,000 U/24 hr). This method appears to be very useful in the diagnosis of pancreatic insufficiency, but it is not sensitive enough to detect lesser degrees of pancreatic damage.

0505 PHARMACOLOGICAL INHIBITION OF DIGESTIVE SECRETIONS IN THE MANAGEMENT OF PANCREATICODUODENAL FISTULAS. (Eng.) Weisz, G. M.; Schramek, A.; Barzilai, A. (Aba Khoushy Sch. Medicine, Technion Israel Inst. Technology, Haifa, Israel). *Int. Surg.* 61(5):279-282; 1976.

0506 ENDOSCOPIC RETROGRADE PANCREATICOGRAPHY AND EXOCRINE PANCREATIC FUNCTION TEST. A COMPARATIVE STUDY [Abstract]. (Eng.) Rolny, P.; Lukes, P.; Gamklou, R.; Nilsson, A. (Sahlgrenska sjukhuset, Univ. Goteborg, Goteborg, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):82; 1977.

0507 NONPANCREATIC TYPE HYPERAMYLASEMIA ASSOCIATED WITH PANCREATIC CANCER. (Eng.) Shimamura, J.; Fridhandler, L.; Berk, J. E. (Dept. Medicine, Univ. California, Irvine, CA). *Am. J. Dig. Dis.* 21(4):340-345; 1976.

0508 ALPHA$_1$ ANTITRYPSIN DEFICIENCY AND PANCREATIC FIBROSIS. (Eng.) Freeman, H. J.; Weinstein, W. M.; Shnitka, T. K.; *et al.* (Dept. Medicine, Univ. Alberta, Edmonton, Canada). *Ann. Intern. Med.* 85(1):73-76; 1976.

0509 10 YEARS' EXPERIENCE TREATING PANCREATIC AND PERIAMPULLARY CANCER. (Eng.) Hines, L. H.; Burns, R. P. (No affiliation given). *Am. Surg.* 42(6):441-447; 1976.

0510 PHYSIOPATHOLOGIC STUDY OF THE ZOLLINGER ELLISON SYNDROME. (Fre.) Gorin, J. P. (37 Ave. Lowendal, Paris, France). *J. Chir. (Paris)* 111(1):75-98; 1976.

0511 SELECTIVE CATHETERIZATION OF THE PANCREATIC VEIN FOR RADIOIMMUNOASSAY IN GLUCAGON SECRETING CARCINOMA OF THE PANCREAS. (Eng.) Ingemansson, S.; Lunderquist, A.; Holst, J. (Dept. Surgery, Univ. Lund, Lund, Sweden). *Radiology* 119 (3):555-556; 1976.

0512 ELEVATED SERUM RIBONUCLEASE IN PATIENTS WITH PANCREATIC CANCER. (Eng.) Reddi, K. K.; Holland, J. F. (Mt. Sinai Sch. Medicine, New York, NY 10029). *Proc. Natl. Acad. Sci. U.S.A.* 73(7):2308-2310; 1976.

0513 MISLEADING SGOT (ASPARTATE AMINOTRANSFERASE VALUES IN OBSTRUCTIVE JAUNDICE DUE TO PANCREATIC CARCINOMA). (Eng.) Gregory, P. B.; Cooney, D. P. (Stanford Sch. Medicine, Stanford, CA 94305). *Am. J. Dig. Dis.* 21(6):509-511; 1976.

0514 COMPUTERIZED AXIAL TOMOGRAPHY FOR DIAGNOSIS OF PANCREATIC CANCER. (Eng.) Wiggans, G.; Schein, P. S.; MacDonald, J. S.; *et al.* (Vincent Lombardi Cancer Res. Center, Washington, DC 20007). *Lancet* 2(7979):233-235; 1976.

See also, 0009, 0126, 0274, 0283, 0389, 0527, 0709.

PANCREAS

Pancreatitis

0515 A NEW PATTERN OF PANCREATIC SECRETION (PBS) OBSERVED IN PANCREATIC EDEMA. (Eng.) Tuzhilin, S. A.; Dreiling, D. A. (Mt. Sinai Medical Sch., City Univ. New York, 100th St. and Fifth Ave., New York, NY 10029). *Am. J. Gastroenterol.* 67(3):220-228; 1977.

Exocrine pancreatic secretion studies were done on 53 patients with pancreatic edema and 3 Thomas cannula dogs to elucidate the pathogenesis of proximal block of pancreatic secretion (P.B.S.). Pancreatic secretion was investigated using secretin and intraduodenal HCl as stimuli. An abnormal pattern of secretion following HCl administration in pancreatic edema patients (20) was observed: there was a significant decrease in flow ($p<0.001$), decreased bicarbonate concentration ($p<0.05$ after 20- to 40-min stimulation), and increased amylase concentration ($p<0.01$-0.05). Reversal of the secretory pattern after i.v. secretin administration (1 clinical U/kg) towards normal for flow and bicarbonate concentration was seen, though the hyperconcentration of the enzyme ($p<0.01$-0.001) compared to the normal patients (13) persisted. Comparison of the weak HCl and strong secretin stimulus to pancreatic secretion in the pancreatic edema patients augmented the abnormal discordant secretory defect (diminution in flow, $p<0.001$); there was an insignificant effect on bicarbonate concentration and an insignificant increase in amylase concentration. This

reversal of the secretory pattern of P.B.S. is due to a larger secretory flow nullifying the dilution effect of interstitial fluid exudation into the duct system. Dehydration with diuretics in pancreatic edema patients (14) reversed the secretory abnormality (P.B.S.). Secretin test response in dogs before and after adrenalin infusion (0.1-1%) showed decreased flow ($p<0.001 \rightarrow 0.10$) and increased enzyme concentration ($p<0.01$). The combined low stimulus-high stimulus approach is suggested to study pancreatic damage or dysfunction.

0516 ENDOSCOPIC RETROGRADE PANCREATOGRAPHY AND SECRETIN-PANCREOZYMIN TEST IN DIAGNOSIS OF CHRONIC PANCREATITIS: A COMPARATIVE EVALUATION. (Eng.) Dobrilla, G.; Fratton, A.; Valentini, M.; Vantini, I.; Cavallini, G.; Angelini, G.; *et al.* (Ospedale Generale Regionale, I-39100 Bolzano, Italy). *Endoscopy* 8(3):118-123; 1976.

The diagnostic values of endoscopic retrograde pancreatography (ERP) and secretin-pancreozymin (S-CCK.PZ) test were compared in 23 patients, 6 with suspected chronic pancreatitis and 17 with proven chronic pancreatitis. The results showed a good correlation between ERP examinations and S-CCK.PZ test only for patients with proven chronic pancreatitis. In patients with suspected chronic pancreatitis, there were discrepancies between ERP (normal results) and S-CCK.PZ test (abnormal for the only parameter "low flow"). The principal types of ERP abnormality were alternate dilatation and stenosis of Wirsung's duct (75%), cystic dilatation of branches (58%), irregular distribution of the branches (41%), and calculi in Wirsung's duct (34%). The time of elimination of the contrast medium was significantly correlated with the S-CCK.PZ test and with the pancreatographic picture.

0517 ULTRASOUND IN THE MANAGEMENT OF ACUTE PANCREATITIS. (Eng.) Duncan, J. G.; Imrie, C. W.; Blumgart, L. H. (Royal Infirmary, Glasgow, G40SF, Scotland). *Br. J. Radiol.* 49(586):858-862; 1976.

Following a proven attack of acute pancreatitis, 38 patients were referred for ultrasound examination when signs or symptoms suggested the development of a pancreatic pseudocyst. Pseudocysts were diagnosed by ultrasound in 23 cases (12 related to alcohol, 6 to biliary disease, 2 to trauma, and 3 to other causes). Five patients had multilocular cysts, four of which were shown by ultrasound to be communicating. Pseudocyst was confirmed in 13 of 14 patients undergoing laparotomy, and surgical drainage was performed in 12 of these patients. Three patients had small cysts (less than 6 cm in diameter) in the head of the pancreas; two were proven at laparotomy and were successfully treated by surgical drainage. The remaining nine cases (8 single cysts and 1 multiple cyst) were monitored over periods of 4-12 weeks and showed spontaneous regression. An area of irregular absorption of ultrasound in 12 additional patients among the 38 examined was interpreted as an inflammatory mass that was confirmed in all 3 cases requiring laparotomy. Serial scanning of these 12 patients showed resolution of the mass over periods of 4-20 weeks. None of these cases developed a pseudocyst, but three required operation, one for an abscess, one for necrosis of the body and tail of the pancreas, and a third for severe pancreatic fibrosis. Three of the 38 patients examined showed no ultrasound evidence of inflammatory mass or cyst, and these 3 improved spontaneously without surgery. Ultrasonic scanning is a useful noninvasive means for distinguishing between pseudocysts and inflammatory masses and for demonstrating communication between multiple cysts.

0518 CALCIFIC PANCREATITIS AND IMMUNOLOGY. (Ger.) Klemm, G.; Rohde, L. (Abteilung fur Poliklinische Diagnostik, Wilhelm-Pieck-Universitat Rostock, Ernst-Heydemann-Str., DDR-25 Rostock, E. Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 36(2): 73-75; 1976.

No change in plasma amylase levels was noted in 24 rats with chronic interstitial calcifying pancreatitis induced by vitamin D_2 (1 mg/100 g, s.c.) and iron dextran (0.3 ml in the pancreatic stroma). Eight animals died of calcifying pancreatitis within 3 months, and body weight decreased on the average by one-third during the first test week. Histologically, chronic granulating inflammation with foreign giant cells as well as pseudoductular parenchyma regeneration were found in the interstitial calcification. Other test results have shown that not only a macromolecule, such as iron dextran, but also isolated rat peritoneal mast cells with IgE can cause degranulation. In calcifying pancreatitis, a mast cell reaction induced by an immune mechanism reaction may be possible.

0519 STUDIES ON SERUM AMYLASE IN NORMAL MAN AND IN ACUTE PANCREATITIS. (Eng.) MacGregor, I. L.; Zakim, D. (Veterans Admin. Hosp., 4150 Clement St., San Francisco, CA 94121). *Aust. N.Z. J. Med.* 6(6):551-556; 1976.

To assess the potential significance of amylase isoenzymes in the diagnosis of pancreatic disease, amylase isoenzyme patterns were determined in human serum, urine, saliva, jejunal juice, and pancreatic tissue. The isoenzymes were separated by isoelectric focusing. The predominant amylase isoenzyme fraction in 11 of 14 samples of autopsy pancreases focused with an isoelectric point between 6.8 and 7.1. The crude homogenate of all such glands that were tested had the ability to hydrolyze *p*-tosyl arginine methyl ester hydrochloride (TAME). TAME esterase activity was not detectable in the crude homogenates of three pancreases with isoamylases focusing with isoelectric points between 6.2 and 6.3, although a number of glands without detectable TAME esterase activity contained isoamylases focusing predominantly at pH 6.8 to 7.1. The amylase isoenzymes in freshly expectorated saliva showed two major isoenzyme peaks with isoelectric points between 5.8 to 6.0, and 6.3 to 6.4. Amylase isoenzymes in aspirates from the

small intestine demonstrated isoenzymes derived from the pancreas and salivary glands. The pattern of isoenzymes of amylase in the sera of normal subjects revealed fractions likely to be derived from the salivary glands and the pancreas. In the majority of sera, the major amylase isoenzyme had an isoelectric point between 5.8 and 6.0. This peak coincided with the major peak from saliva, and was therefore of salivary gland origin. The pattern of isoenzymes in sera and urine from patients with acute pancreatitis differed quantitatively from that in normal subjects. The predominant peak of amylase activity in acute pancreatitis sera had an isoelectric point of 6.8 to 7.1. The activity of the amylase isoenzymes of presumed salivary origin was also increased. The mean renal clearance of amylase as the percentage of creatinine clearance in four patients with acute pancreatitis was 4.7 ± 1.2. The clearance of the pancreatic peak with an isoelectric point of 6.8 to 7.0 was 7.1 ± 3.5%, and the clearance of the salivary peak with an isoelectric point between 5.8 and 6.0 was 3.0 ± 1.1%. The increased renal clearance of amylase in acute pancreatitis may be due partly to the increased proportion of the smaller molecular weight pancreatic amylase. However, the increase in the renal clearance of salivary amylase suggests that the kidneys are also involved. These studies also indicate that the majority of amylase activity in normal sera is of salivary gland origin.

0520 POSSIBLE MECHANISMS OF NORMAL AMYLASE ACTIVITY IN HYPERLIPEMIC PANCREATITIS. (Eng.) Mishkin, S.; Bates, J.; O'Hashi, J.; Schneider, P.; Sniderman, A. D.; Wolf, R. O. (Royal Victoria Hosp., 687 Pine Ave. W, Montreal, Quebec H3A 1A1, Canada). *Can. Med. Assoc. J.* 115(10): 1016-1019; 1976.

Possible mechanisms of normal amylase activity in patients with hyperlipemic pancreatitis were investigated. When lipemic serum from three patients with acute pancreatitis and type IV hyperlipemia was fractionated into very-low-density lipoproteins (VLDL) and clear serum amylase activities (determined by the Phadebas method) in the VLDL fractions (20, 40, and 384 IU/l, resp.) and clear serum fractions (67, 157, and 525 IU/l, resp.) never exceeded the amylase activities in the original lipemic sera (165, 490, and 1,875 IU/l, resp.). This fact indicates that the removal of lipid turbidity from the original lipemic sera did not unmask high levels of amylase activity in the remaining clear serum. The addition of the VLDL and clear serum fractions or the addition of VLDL and chylomicrons from asymptomatic patients with hyperlipemia to nonlipemic serum from three patients with routine acute pancreatitis and high serum amylase activities (1000-6,000 IU/l) did not inhibit the amylase activities or alter the electrophoretic mobility of amylase isoenzymes. Therefore, the normal amylase activity often observed in hyperlipemic pancreatitis does not result from an inhibition of amylase activity by serum lipoproteins.

0521 POST-TRANSPLANTATION PANCREATITIS. (Eng.) Fernandez, J. A.: Rosenberg, J. C. (Dept. Surgery, Wayne State Univ., Detroit, MI). *Surg. Gynecol. Obstet.* 143(5):795-798; 1976.

A review of the literature on postrenal transplantation pancreatitis is presented along with case reports of two women developing this condition. Post-transplantation pancreatitis occurs with a frequency of about 3% and carries a mortality of 70%. An average of 216 days (range, 1-570 days) following transplantation elapses before the onset of pancreatitis, which occurs most frequently in men in the fourth decade of life. Post-transplantation pancreatitis was unsuspected in 35% of patients with this condition who died and were autopsied. Factors that predispose to pancreatitis in the post-transplant period include immunosuppressive therapy with azathioprine and steroids, secondary hyperparathyroidism, and increased susceptibility to infection. The diagnosis of pancreatitis depends greatly on an increase in the serum amylase level, which had been found to vary from 2 to 40 times the normal value in post-transplant patients with pancreatitis. In one of the two cases reported, the serum amylase level was 340 U/100 ml 3 months post-transplant, and a serum amylase level as high as 1,600 U/100 ml was recorded in the other patient 3.5 months after renal transplantation. After renal transplantation, patients should frequently have serum and urinary amylase determinations, with these determinations being compared to the creatinine clearance rates. The diagnosis of pancreatitis should be considered if any abdominal symptoms occur. It is possible that post-transplant pancreatitis is caused by a virus and is related to the immunosuppressive state. The presence of cytomegalovirus in the pancreas has been reported, but its pathogenetic significance is uncertain.

0522 COMPLEMENT MEDIATED ACINAR CELL NECROSIS IN PANCREATITIS INDUCED BY BASEMENT MEMBRANE ANTIBODIES. (Eng.) Seelig, R.; Seelig, H. P. (Inst. Pathology, Univ. Heidelberg, Heidelberg, W. Germany). *Virchows Arch. [Pathol. Anat.]* 371(1): 69-77; 1976.

0523 ACUTE PANCREATITIS IN TWO CASES OF INFECTIOUS MONONUCLEOSIS. (Eng.) Hedstrom, S. A.; Belfrage, L. (Univ. Hosp., Lund, Sweden). *Scand. J. Infect. Dis.* 8(2):124-126; 1976.

0524 BISALBUMINEMIA IN PANCREATITIS: STRUCTURAL MODIFICATIONS OF HUMAN SERUM ALBUMIN BY PROTEOLYTIC ENZYMES OF THE PANCREAS. (Fre.) Rousseaux, J.; Debeaumont, D.; Scharfman, A.; *et al.* (Fac. Med., Lille, France). *Clin. Chim. Acta* 71(1): 35-46; 1976.

0525 SEQUESTRECTOMY AND HYPERALIMENTATION IN THE TREATMENT OF HEMORRHAGIC PANCREATITIS. (Eng.) White, T. T.; Heimbach, D. M. (Univ. Washing-

ton Sch. Medicine, Seattle, WA 98195). *Am. J. Surg.* 132(2):270-275; 1976.

0526 THE VALUE OF ALKALINE PHOSPHATASE (AP) AS A MARKER FOR COMMON BILE DUCT (CBD) STENOSIS IN ALCOHOLIC PANCREATITIS. (Eng.) Littenberg, G.; Afroudakis, A.; Kaplowitz, N. (Wadsworth Veterans Admin. Hosp., Los Angeles, CA). *Gastroenterology* 73(5):1232; 1977.

0527 PAROTID GLAND FUNCTION IN PATIENTS WITH PANCREATIC DISEASE [Abstract]. (Eng.) McColl, K. E. L.; Brodie, M. J.; White-Smith, R.; Thomson, T. J. (Stobhill Hosp., Glasgow, Scotland). *Gut* 18(11):A960; 1977.

0528 SINGLE CENTRE DOUBLE-BLIND TRIAL OF TRASYLOL THERAPY IN PRIMARY ACUTE PANCREATITIS [Abstract]. (Eng.) Imrie, C. W.; Benjamin, I. S.; Ferguson, J. C.; Thomson, W. O.; McKay, A. J.; O'Neil, J.; Blumgart, L. H. (Royal Infirmary, Glasgow, Scotland). *Gut* 18(11):A957-A958; 1977.

0529 VALUE OF DIAGNOSTIC PERITONEAL LAVAGE TO DETERMINE THE SEVERITY OF ACUTE PANCREATITIS [Abstract]. (Eng.) McMahon, M. J.; Blackett, R. L.; Pickford, I. (General Infirmary, Leeds, England). *Gut* 18(11):A958; 1977.

0530 ERCP AND SURGERY IN RECURRENT ACUTE PANCREATITIS [Abstract]. (Eng.) Cotton, P. B.; Croker, J. R. (Middlesex Hosp., London, England). *Gut* 18(11):A941; 1977.

0531 GIP CELLS IN CHRONIC PANCREATITIS [Abstract]. (Eng.) Buchan, A. M. J.; Polak, J. M.; Bloom, S. R.; Besterman, H.; South, M.; McCloy, J. H.; *et al.* (Royal Postgraduate Medical Sch., London, England). *Gut* 18(11):A983; 1977.

0532 Mycoplasma pneumoniae--AN AETIOLOGICAL FACTOR IN ACUTE PANCREATITIS [Abstract]? (Eng.) Freeman, R.; McMahon, M. J. (Chapel Allerton Hosp., Leeds, England). *Gut* 18(11):A981; 1977.

0533 PAIRED PANCREATIC FUNCTION STUDIES USING PURE PANCREATIC JUICE AND DUODENAL JUICE [Abstract]. (Eng.) Denyer, M. E.; Cotton, P. B.; Kyne, M.; Miller, A. (Middlesex Hosp. and Medical Sch., London, England). *Gut* 18(11):A980; 1977.

See also, 0119, 0121, 0127, 0260, 0504, 0512, 0652.

LIVER AND BILIARY TRACT

0534 LAPAROSCOPY WITH PARTICULAR REFERENCE TO MANAGEMENT OF DISEASES OF THE LIVER. (Eng.) Walker, R. J.; Williams, R. *In: Modern Topics in Gastrointestinal Endoscopy.* Edited by K. F. Schiller, and P. R. Salmon. (Chicago: William Heinemann Medical Books): 275-295; 1976.

In this review, laparoscopy is discussed in terms of equipment, technique, indications for use, results, and complications. The normal and pathological appearance of abdominal organs observed by laparoscopy is described. Biopsy is considered to be an essential part of the technique and should be performed, if possible, to confirm laparoscopic findings. The most common indications for the procedure are hepatomegaly, unexplained liver function test abnormalities, suspected cirrhosis or tumor, jaundice, and ascites. The correct diagnosis was made by laparoscopy in 75 of 100 consecutive patients examined for the aforementioned indications. Five case histories are presented to illustrate typical diagnostic situations in which laparoscopy proved to be valuable.

0535 DISPOSITION OF AMINOPYRINE, ANTIPYRINE, DIAZEPAM, AND INDOCYANINE GREEN IN PATIENTS WITH LIVER DISEASE OR ON ANTICONVULSANT DRUG THERAPY: DIAZEPAM BREATH TEST AND CORRELATIONS IN DRUG ELIMINATION. (Eng.) Hepner, G. W.; Vesell, E. S.; Lipton, A.; Harvey, H. A.; Wilkinson, G. R.; Schenker, S. (Harbor General Hosp., Torrance, CA 90509). *J. Lab. Clin. Med.* 90(3):440-456; 1977.

The extent of hepatic elimination of various drugs was compared in controls, patients receiving anticonvulsants that stimulate the microsomal oxidizing system, and patients with hepatobiliary diseases (hepatocellular disease, hepatic neoplasm, or cholestasis). Aminopyrine metabolism was measured by the aminopyrine breath test, diazepam elimination by a newly developed ^{14}C-diazepam breath test (DBT) and by conventional plasma diazepam measurements, antipyrine clearance by drug analysis in saliva, and indocyanine green (ICG) clearance by its removal rate from plasma. The DBT correlated well with diazepam half-life when all subject groups were combined ($r=-0.65$, $p<0.001$). Anticonvulsant drugs

enhanced the elimination of aminopyrine, diazepam, and antipyrine, but the degree of stimulation was significantly lower for aminopyrine; ICG elimination was not altered by anticonvulsant drugs. The mean elimination for each drug tested in patients with hepatocellular and neoplastic liver diseases was depressed significantly and to a similar extent. In cholestasis, elimination of antipyrine and ICG but not of aminopyrine or diazepam was depressed significantly. Finally, there was no consistent correlation of the elimination of the four drugs tested in controls alone, and the correlation, although sometimes statistically significant, was only marginal (r=0.30-0.67) in the combined hepatobiliary group alone. Attempts to extrapolate pharmacokinetic data from one drug to another in individual subjects may be misleading.

0536 GROWTH HORMONE AND PROLACTIN RESPONSES TO THYROTROPIN-RELEASING HORMONE IN PATIENTS WITH SEVERE LIVER DISEASE. (Eng.) Panerai, A. E.; Salerno, F.; Manneschi, M.; Cocchi, D.; Muller, E. E. (Dept. Pharmacology, Univ. Milan, Milan 20129, Italy). *J. Clin. Endocrinol. Metab.* 45(1):134-140; 1977.

To determine whether abnormalities in growth hormone release (GH) in severe liver diseases are related to a positive GH response to thyrotropin-1 releasing hormone (TRH, 400 µg) was administered i.v. to 10 patients with severe liver disease and to 10 controls. TRH induced a clear-cut GH rise (≥10 ng/ml) in seven patients; this occurred 15-120 min after the injection. TRH had no effect on GH levels in controls. The mean baseline GH levels were higher in patients than in controls (3.8 ± 1.0 versus 0.8 ± 0.4 ng/ml, $p<0.02$). An exaggerated and sustained increase in prolactin (PRL) was present after TRH in the subjects with liver disease, whose mean baseline plasma PRL levels were within the normal range. Although the rise in GH induced by TRH may be due primarily to a disturbance in the neuroendocrine control of GH secretion, the exaggerated PRL rise after the tripeptide seems to be caused by peripheral factors.

0537 METHODS FOR DETECTION OF ANTI-ALBUMIN AUTOANTIBODIES IN HEPATIC DISEASES. (Eng.) Lenkei, R.; Ghetie, V. (Dept. Immunology, Babes Inst., Bucharest 6, 76, 210, Rumania). *J. Immunol. Methods* 16(1):23-30; 1977.

The application of three methods for the detection and quantitation of anti-albumin autoantibodies (AAA) in human sera is described. The methods are based on their reaction with a new antigenic site of glutaraldehyde-polymerized human serum albumin (HSA). The first method is a double diffusion technique (ID-HSA) where the sera to be tested are reacted with a copolymer of glutaraldehyde activated HSA (copoly-HSA). The reaction is estimated by the lowest copoly-HSA concentration giving precipitation with the undiluted sera. The second method is based on agglutination (HA) by AAA-positive sera of human RBC coated with glutaraldehyde-polymerized HSA (EPA). The reaction is quantified as the highest dilution of sera giving agglutination. The third method uses glutaraldehyde-insolubilized HSA as an immunoabsorbent able to bind AAA in positive sera. The quantity of protein absorbed, expressed in µg antibody/ml serum, measures the level of AAA in sera. Glutaraldehyde treatment of HSA led to the formation of soluble polymers that were able to react strongly in immunodiffusion with patients' reference sera. The HSA polymers responsible for this reaction could be prepared in high yield; more than 85% of the treated HSA was polymerized. At pH 9.0, glutaraldehyde-activated HSA bound a further amount of nontreated HSA yielding copoly-HSA. The correlation between the immunodiffusion test with animal sera (ID-AS) and ID-HSA was investigated in sera from 86 patients with acute viral hepatitis, 97 blood donors, and 84 contacts for hepatitis B surface antigen. Agreement between results occurred in 89.6% of the sera from the hepatitis patients, 84.2% of the sera from blood donors, and 91.3% of the sera of the hepatitis contacts. HA-EPA tests in the sera of healthy donors and patients with liver diseases showed that titers up to 1:32 did not indicate liver dysfunction. Titers of 1:64 to 1:4,096 were recorded in the sera of patients with liver disease and were significantly correlated with positive ID-HSA tests in viral hepatitis and in HBsAg contacts. The immunoabsorbent technique permitted the estimation of AAA concentrations in normal and pathological sera. Generally, a lack of reaction with copoly-HSA in immunodiffusion was associated with an AAA concentration below 100 µg/ml, while higher concentrations of AAA (>200 µg/ml) were associated with precipitation of copoly-HSA at 39-625 µg/ml. Highly significant correlation coefficients were calculated between the immunoabsorbent technique, ID-HSA, and HA-EPA techniques.

0538 SERUM IMMUNOGLOBULINS AND ORGAN NON-SPECIFIC ANTIBODIES IN DISEASES OF THE LIVER. (Eng.) Husby, G.; Skrede, S.; Blomhoff, J. P.; Jacobsen, C. D.; Berg, K.; Gjone, E. (Rikshospitalet, Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(3):297-304; 1977.

Serum immunoglobulins and complement fraction 3 (C3) levels, rheumatoid factors (RF), hepatitis B surface antigen (HB antigen) and antibody (HB antibody), and auto-antibodies to smooth muscle (SMA), mitochondria (MA), and nuclei (ANA) were determined in 202 patients with one of nine liver diseases. Hypergammaglobulinemia was a prominent feature in most groups, IgG being particularly raised in active chronic hepatitis, IgM in primary biliary cirrhosis, and IgA in alcoholic liver disease, resp. IgE was often increased in alcoholic liver disease and was frequently low in hepatic tumors, whereas IgD showed no typical pattern in any liver disorder. SMA was most frequently found in active chronic hepatitis (68%), and MA in primary biliary cirrhosis (58%), while ANA was detected in 50% of the patients with active chronic hepatitis. However, a pronounced over-lap of tissue antibodies was observed among the various groups of liver disease, particularly in active chronic hepatitis and primary biliary

cirrhosis. The concurrent presence of SMA and ANA was most frequent in active chronic hepatitis. Antibody titers might have provided better diagnostic discrimination, since titration of antibodies was not performed. Low C3 levels in active chronic hepatitis were correlated with low levels of other liver synthesized proteins, and no evidence was found on increased consumption by immunologic reactions.

0539 THE ASSOCIATION OF FAMILIAL LIVER DISEASE, SUBEPIDERMAL IMMUNOPROTEINS, AND MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS. (Eng.) Dobrin, R. S.; Hoyer, J. R.; Nevins, T. E.; Sharp, H.; Gentry, W. C.; Vernier, R. L. (Children's Hosp. Medical Center of Northern California, 51 and Grove St., Oakland, CA 94609). *J. Pediatr.* 90 (6):901-909; 1977.

A new form of congenital hepatic disease is reported. Each of the four siblings had splenomegaly, hypersplenism, a small liver, biochemical evidence of hepatic excretory dysfunction and hepatocellular damage, kidneys without demonstrable cysts, and normal blood pressure. An evaluation of serum immunoproteins, autoantibodies, histocompatibility antigens, and mixed lymphocyte reactivity further defined the immunologic features of this syndrome. Extrahepatic manifestations included a papulosquamous dermatitis with deposition of immunoglobulins and complement in both normal and abnormal skin; a membranoproliferative glomerulonephritis with subendothelial deposits; arthritis; and pericardial, pleural, and synovial effusions. Further evaluation of this family is required to better understand the pathogenesis of this disease.

0540 CRYPTOGENIC LIVER ABSCESS. (Ger.) Gunther, B.; Gruner, A.; Bull, U. (Chirurgische Klinik der Universitat, Nussbaumstrasse 20, D-8000 Munich 2, W. Germany). *Munch. Med. Wochenschr.* 119(9):305-308; 1977.

Cryptogenic liver abscess was diagnosed in a 61-yr-old woman and a 50-yr-old man by liver scintigraphy and celiacography (in one case). Both patients had fever and were in severely impaired general condition, but there were no pathognomonic symptoms. After aspiration of the purulent liquid, transperitoneal drainage was applied. The cavity was irrigated daily with saline solution containing antibiotics and enzyme preparations. The treatment was successful, both patients were in good condition at discharge and at the follow-up several months later, at which time no filling defects were seen in the liver scintigraphically. The findings indicate that transperitoneal open drainage is the method of choice in the treatment of patients with cryptogenic liver abscess.

0541 PROGNOSIS OF HEPATOPATHY IN HOMOZYGOUS ALPHA-1-ANTITRYPSIN DEFICIENCY. (Ger.) Karitzky, D.; Kunzer, W. (Universitats-Kinderklinik, D-7600 Freiburg i. Br., W. Germany). *Monatsschr. Kinderheilkd.* 125(5):472-473; 1977.

The prognosis of children with hepatopathy with homozygous alpha$_1$-antitrypsin deficiency (aATD) of the protease inhibitor type ZZ is discussed on the basis of the observation of 10 cases. The aATD was detected at the age of 3-8 weeks in connection with jaundice, hepatomegaly, or dystrophy in five cases, and serendipitously in five others. In the latter group, only one child developed progressive liver cirrhosis. In four of the other five patients, in whom aATD was diagnosed at an early age in connection with liver cirrhosis, bile duct hypoplasia, infectious hepatitis, or liver fibrosis, the initially highly elevated transaminase levels and other clinical findings normalized during the follow-up period (6-15 yr). The aATD was subject to fluctuations, and its increase was accompanied by the normalization of the transaminase level. The findings indicate that the normalization of the transaminase levels by the age of 3-5 yr is prognostically favorable, and that the frequency of juvenile cirrhosis in children with aATD is lower than the 20-30% it is generally believed to be. Persons with homozygous aATD are not overrepresented among patients with liver cirrhosis.

0542 ALPHA-FETOPROTEIN AND THE DIAGNOSIS OF LIVER CELL CANCER. (Eng.) Purves, L. R. *In: Liver Cell Cancer.* Edited by H. M. Cameron, D. A. Linsell, and G. P. Warwick. (New York: Elsevier Scientific Publishing Company): 61-79; 1976.

The physiology of alpha fetoprotein (AFP) is described, and its use in screening for liver cell carcinoma is reviewed. The use of AFP in the diagnosis of liver cell cancer is invaluable; most cases can be accurately diagnosed or justification can be provided to pursue the diagnosis by other means. Whether the measurement of AFP is justified as an epidemiological tool is doubtful, since there is a normal AFP level (the effects of subclinical hepatitis altered this normal level) and since liver cell cancer is, in general, not responsive to treatment. Further prospective surveys using radioimmunoassay for the quantitation of AFP are necessary before it can be used to detect evolving cancer at an early enough stage to be beneficial to the patient.

0543 THERAPEUTIC INDICATIONS FOR CHILDREN WITH PRIMARY LIVER TUMORS. (Fre.) Mitrofanoff, P. (No affiliation given). *Ann. Chir. Infant.* 17(5/6):479-483; 1976.

The types of therapy for children with primary liver tumors are reviewed. Surgery (tentative hepatectomy) is recommended for malignant tumors, as radiotherapy or chemotherapy is not very successful. However, radiotherapy (up to 1,000 rads) is indicated for stopping the involution of angioma, and, in serious cases, ligature of the hepatic artery has been effective. Corticotherapy has also been used for fast-growing angiomas. Surgery has been used

for cystic lymphangiomas. Angiomas usually occur in the first 3 months of life, but malignant tumors usually develop later. Malignancies can be detected by clinical and biological tests and radiological vascular exploration. Determination of α-fetoprotein levels is especially useful in distinguishing between angiomas and hamartomas in young infants. Surgery for angioma has proved fatal in some cases: 6/17 children died after excision, but 3/3 given radiotherapy were cured. A review of 81 children with malignant tumors (hepatoblastoma/hepatocarcinomas and sarcoma) showed that 68 had died, compared to 89/155 with benign tumors (angioma and hamartoma).

0544 THE TREATMENT OF LIVER CELL CANCER. (Eng.) Falkson, G. *In: Liver Cell Cancer*. Edited by H. M. Cameron, D. A. Linsell, and G. P. Warwick. (New York: Elsevier Scientific Publishing Company): 81-92; 1976.

Surgery, radiotherapy, and chemotherapy of liver cell cancer are reviewed. Primary liver cell cancer is seldom resectable, and is not radiosensitive. Clinical control must be sought by chemotherapy. 5-Fluorouracil given intravenously has been generally accepted in the U.S.A., but worthwhile responses have not followed extensive clinical trial of this agent in Bantu patients. Alkylating agents, antimetabolites, antibiotics, and other drugs have been carefully evaluated in patients with liver cell cancer and have been found not to be of particular value. Recent advances in chemotherapy make it more likely that treatment with cytostatic agents will lead to better results.

0545 A PHASE II STUDY OF ADRIAMYCIN (NSC 123127) IN PATIENTS WITH HEPATOCELLULAR CARCINOMA FROM ZAMBIA AND THE UNITED STATES. (Eng.) Vogel, C. L.; Bayley, A. C.; Brooker, R. J.; Anthony, P. P.; Ziegler, J. L. (Univ. Miami, Sch. Medicine, 1400 N.W. 10th Ave., Center House--Roof Garden, Miami, FL 33136). *Cancer* 39(5):1923-1929; 1977.

Nineteen Zambian and 22 American patients with hepatocellular carcinoma were treated with adriamycin (ADM; 20-75 mg/m^2, i.v, depending on the initial serum bilirubin level) every 3 weeks. Four of 16 "good risk" Zambian and American patients (those eligible for 75 mg of drug) had objective responses, while in 5 others there was evidence of either transient tumor regression or disease stabilization; only 3 of 25 "poor risk" patients who received 20-60 mg/m^2 had objective responses. Even in this latter group, however, transient objective signs of tumor regression were noted in four patients. Toxicity at 75 mg/m^2 was more profound than with lower doses but was tolerable and reversible. It is concluded that ADM does yield reproducible and objective antitumor responses in hepatocellular carcinoma and that it is worthy of further investigation.

0546 5-FLUOROURACIL IN HEPATOCELLULAR CARCINOMA: REPORT OF TWENTY-ONE CASES. (Eng.) Link, J. S.; Bateman, J. R.; Paroly, W. S.; Durkin, W. J.; Peters, R. L. (Univ. Southern California Sch. Medicine, LAC-USC-MC, 1200 N. State St., Los Angeles, CA 90033). *Cancer* 39(5):1936-1939; 1977.

Twenty-one patients with biopsy-proven hepatocellular carcinoma were treated with oral (group 1) or intravenous (group 2) 5-fluorouracil (15 mg/kg/week for a period of 6 weeks) according to a randomized treatment schedule. There were no objective responses in either treatment group. One patient on oral therapy showed a transient 25% tumor reduction, and another patient showed stable disease for 12 weeks after the beginning of the therapy. Overall response in the study was zero percent, but two patients did receive marginal therapeutic benefit. The median survival for the group was 65 days. The median survival for orally treated patients was 72 days compared to 45 days for the group treated with intravenous 5-fluorouracil ($p<0.20$). Toxicity occurred in 46% of patients treated, but was tolerable; mild gastrointestinal toxicity was the most commonly noted form, occurring in 30% of patients. Marrow suppression occurred in 18%, but with no severe episodes. It is suggested that weekly oral or intravenous 5-fluorouracil administration has a minimal role in the treatment of hepatocellular carcinoma.

0547 THE GEOGRAPHIC PATHOLOGY OF LIVER CELL CANCER. (Eng.) Linsell, D. A.; Higginson, J. *In: Liver Cell Cancer*. Edited by H. M. Cameron, D. A. Linsell, and G. P. Warwick. (New York: Elsevier Scientific Publishing Company): 1-16; 1976.

The frequency and types of liver cancers observed in different areas of the world are reviewed. Liver cancer remains a major problem of many areas of the developing world and is probably one of the most frequent cancers in man. Although there is some evidence that the incidence may be falling in Africa, there are reports of a higher incidence in Europe. The association of the cancer with cirrhosis is well established, but the precise sequence of events is still unclear. One type of cirrhosis, the macronodular, is more frequently associated with the cancer, and this is the cirrhosis most commonly observed in countries reporting high frequencies of liver cell cancer. It is now possible to evaluate some of the environmental factors, both viral and chemical, that may be involved in this entity's increased frequency.

0548 THE CLINICAL PROBLEM OF LIVER CELL CANCER IN A HIGH INCIDENCE AREA. (Eng.) Bagshawe, A.; Cameron, H. M. *In: Liver Cell Cancer*. Edited by H. M. Cameron, D. A. Linsell, and G. P. Warwick. (New York: Elsevier Scientific Publishing Company): 45-59; 1976.

The clinical features of liver cell cancer in developing countries are described and compared with those seen in Europe and America. In the latter

countries, it affects older people, is often superimposed on recognized cirrhosis, and produces a less dramatic illness. The disease must be distinguished from liver abscess, parasitic liver diseases, and cirrhosis, as well as from secondary liver tumors; the last are relatively infrequent in most areas with a high incidence of liver cell cancer. Final proof of the diagnosis rests on the histology of the tumor, but the elevation of serum alpha fetoprotein to a level detectable by immunodiffusion is seldom seen, except in the liver cell cancer, pregnancy, and certain teratomas. More sensitive methods of measuring alpha fetoprotein may give false positive results. Hepatic scintigraphy and angiography allow accurate tumor localization for biopsy and will determine its extent and vascularization if surgical resection or intraarterial perfusion of chemotherapeutic agents is planned. However, the results of chemotherapy are so poor that there is seldom justification for subjecting patients to the toxicity of conventional cytotoxic drugs; management usually consists of the relief of pain and sympathetic terminal care.

0549 THE BACKGROUND TO LIVER CELL CANCER. (Eng.) Anthony, P. P. *In: Liver Cell Cancer*. Edited by H. M. Cameron, D. A. Linsell, and G. P. Warwick. (New York: Elsevier Scientific Publishing Company): 93-120; 1976.

Liver diseases, occurring in geographical areas where hepatocellular carcinoma is common, and factors thought to be involved in the genesis of cirrhosis are reviewed. Liver cell cancer occurs most frequently in cirrhotic liver, and it seems that almost any disease process that causes cirrhosis is associated with an increased risk of malignancy. The magnitude of this risk varies throughout the world, and it may be determined by differences in etiology. Race, sex, and genetics have little role to play. Alcoholism and, possibly, malnutrition have a potentiating influence, the nature of which is unclear. Parasites are not related to liver cell carcinoma, but infestation with various flukes, notably *Clonorchis sinesis*, is associated with a high incidence of intrahepatic bile duct carcinoma in South East Asia. Naturally occurring carcinogens, particularly aflatoxins, and the hepatitis B virus may be responsible for the majority of liver cell carcinoma in the tropics and subtropics. Liver cell cancer, in all its forms, remains a relatively rare disease in western countries, but its incidence is slowly rising and may be further increased by newly discovered, though yet unproven, carcinogens such as gonadal steroids and vinyl chloride monomer.

0550 PRIMARY HEPATOCELLULAR CARCINOMA--A RESUME. (Eng.) Rubin, E. *In: Liver Cell Cancer*. Edited by H. M. Cameron, D. A. Linsell, and G. P. Warwick. (New York: Elsevier Scientific Publishing Company): 279-286; 1976.

Factors involved in the study of hepatocellular carcinoma are reviewed. The origin and nature of primary hepatocellular carcinoma remains an enigma, but a number of factors render the tumor an excellent model for the study of neoplasms. These include world-wide incidence, geographic distribution, putative association with toxins, the high incidence of production of embryonal proteins, and the ease of production of the cancer in experimental animals. The challenges for the future include a better understanding of the reasons underlying the geographic distribution, the relationship of tumor development to cirrhosis and hyperplastic nodules, the multicentric nature of the tumor, the induction of tumors by chemicals in experimental animals and modifying factors, and the immunologic aspects of primary hepatic carcinoma.

0551 EARLY LESIONS AND DEVELOPMENT OF PRIMARY HEPATOCELLULAR CARCINOMA IN MAN--ASSOCIATION WITH HEPATITIS B VIRAL INFECTION. (Eng.) Okita, K.; Kodama, T.; Harada, T.; Noda, K.; Fukumoto, Y.; Takenami, T.; *et al.* (Yamaguchi Univ., Sch. Medicine, Ube City, Yamaguchi Prefecture 755, Japan). *Gastroenterol. Jpn.* 12(2):51-57; 1977.

Serum and liver biopsy specimens from 103 patients with chronic liver disease were examined for an association between serum α-fetoprotein (AFP) and liver cell dysplasia, which is postulated to be a precursor of primary hepatocellular carcinoma. Among 58 cases of chronic active hepatitis, 14 showed liver cell dysplasia, and 71.4% of these 14 cases had a serum AFP level of >200 ng/ml. Of the remaining 44 cases, 43.1% had a serum AFP level of >200 ng/ml. Liver cell dysplasia was also seen in 13/55 cases of liver cirrhosis, and 69.2% of these 13 cases had a serum AFP level of >200 ng/ml; this level was found in only 30.9% of the remaining 42 cases. Direct fluorescence antibody studies of 20 patients with high serum AFP levels indicated a specific localization of AFP in the dysplastic cells. Hepatitis B-associated DNA polymerase assays revealed a significantly high level of DNA polymerase activity (mean, about 1,800 counts/min) in the sera of patients with chronic active hepatitis, compared with a background level of 900 counts/min for normal subjects.

0552 TREATMENT OF VARIOUS FORMS OF ICTERUS OR CHOLESTASIS WITH PHENOBARBITAL. (Ger.) Stiehl, A. (Med. Univ.-Klinik Heidelberg, Bergheimer Strasse 59, 6900 Heidelberg, W. Germany). *Z. Gastroenterol.* 15(6):404-416; 1977.

The results of the treatment of various forms of icterus or cholestasis with phenobarbital (PB) are reviewed. In the liver, PB induces an increase in the endoplasmic reticulum, an increase of cytoplasmic proteins, and induction of microsomal enzymes such as bilirubin (BL) glucuronyl transferase, which catalyzes the conjugation of BL. PB also increases bile flow, and it improves BL conjugation in patients with Crigler-Najjar syndrome, Gilbert-Meulengracht disease, or newborn icterus. In patients with insufficient BL excretion, such as those with Dubin-Johnson disease, PB caused a decrease in serum BL levels by increasing biliary BL excretion. In

cholestasis, PB caused an increase in biliary excretion of bile-binding substances, leading to a decrease in icterus, pruritus, and hyperlipemia. Daily therapy with PB (120-300 mg) improved icterus and pruritus in the majority of patients, but it was discontinued in some due to heavy sedation. In addition to improving icterus and pruritus, PB has also been observed to produce a marked improvement in the plasma clearance of rose bengal and cholestyramine. Side-effects attributed to PB include a rise in alkaline phosphatase activity and CNS and inflammatory symptoms in newborns.

0553 PURPURA CRYOMACROGLOBULINEMIA IN TRANSIENT HEPATIC PORPHYRIA WITH LIVER CIRRHOSIS. (Ger.) Simon, N.; Husz, S.; Masszi, J. (Univ.-Hautklinik, Postfach 480, H-6701 Szeged, Hungary). *Hautarzt* 28(6):303-307; 1977.

A 49-yr-old man with porphyria cutanea tarda and histologically verified liver cirrhosis also had purpuric, urticarial, and telangiectasial skin symptoms as well as a mixed IgM/IgG cryomacroglobulinemia for 8 yr. The liver disease was considered the primary affliction, accompanied by secondary cryomacroglobulinemia. Histology of the upper dermis of purpuric and urticarial skin revealed RBC extravasates, perivesicular infiltrates from polymorphous nuclear neutrophilic leukocytes, lymphocytes, fibroblasts, individual plasma cells, and eosinophil granulocytes. Immunofluorescence of purpuric lesions showed homogenous vessel color, many small granular deposits, thrombus formation with anti-human globulin, IgG, IgM, and complement. In urticarial skin, the granular deposits along the papillary vessels were much more marked than in purpuric lesions. Porphyrin was not detected in either skin lesion. Histology of a liver biopsy showed typical signs of liver cirrhosis. The clinical picture and amount of excreted porphyrin were not influenced by repetitive bleedings, and treatment with prednisolone (30 mg/day for 5 months, decreased to 10 mg/day for 3 further months) was only partially effective. However, porphyrin content in urine returned to normal, which could have been due to long-term steroid treatment or to progression of cirrhosis.

0554 ON EOSINOPHILIC HEPATOPATHY IN EOSINOPHILIC RETICULAR HYPERPLASIA OF THE SKIN. (Ger.) Korting, G. W.; Schramm, P. (Hautklinik, Johannes-Gutenberg-Universitat, Langenbeckstrasse 1, D-6500 Mainz, W. Germany). *Arch. Dermatol. Res.* 259(1):43-47; 1977.

Eosinophilic hepatopathy was diagnosed in a 40-yr-old woman with eosinophilic reticular hyperplasia of the skin. The anamnestic data included endogenous eczema at the age of 15 yr, transient jaundice in 1966, and ampicillin-induced exanthema in 1974. A tumor, tentatively diagnosed as eosinophilic granuloma, was removed from the buccal cavity in 1974, at which time 26% eosinophilia of the blood was found. The patient developed nodules in the mandibular region, nodular infiltration in the submandibular, sternal and thoracic regions, and eczema of the hip and hands. The blood eosinophil count was 33%, the sternal bone marrow showed hypereosinophilia, and Australia antigen was found in the serum. The suspect liver scintigram and the pathological serum enzyme levels (glutamic oxaloacetic transaminase, 76 mU/ml; glutamic-pyruvic transaminase, 187 mU/ml; gamma-glutamyl transaminase, 49 mU/ml) prompted laparoscopy and liver biopsy, which revealed HBsAg-positive chronic persistent hepatitis, proliferation of fibers in the portal region, and focal clusters of eosinophil cells (eosinophilic hepatopathy). The findings indicate that, contrary to current belief, the involvement of internal organs, particularly of the liver, in reticular hyperplasia of the skin seems to be possible.

0555 REYE'S SYNDROME. (Ger.) Wehinger, H. (Universitats-Kinderklinik Freiburg i. Br., W. Germany). *Klin. Paediatr.* 189(3):206-210; 1977.

The clinical symptoms, diagnosis, etiology, pathogenesis, and therapy of Reye's syndrome are presented. The condition is an acute and often fatal childhood syndrome of encephalopathy and fatty degeneration of the liver, marked by rapid development of brain swelling and hepatomegaly and by disturbed consciousness and seizures. The smooth endoplasmic reticulum shows a proliferation and an increase of peroxisomes, and the glycogen content of the endoplasmic reticulum decreases with severity of the disease. The disease is more predominant in girls, and in the majority of cases it is preceded by influenza B or varicella infection. Elevated ammonia and creatinine phosphokinase levels are also common, as is mitochondrial damage. The early disease stages are characterized by lethargy, delirium, and intermittent coma, whereas decerebral symptoms and muscle hypotony precede death. Intensive monitoring of electrolytes, acid-base status, serum osmolarity, and blood sugar is recommended. Therapy with neomycin, citrullin, vitamin K, glucose, and insulin is warranted for all stages. When cerebral edema is present, Mannit and dexamethasone should be given; intubation is required for respiratory alkalosis. Phenobarbital and diphenylhydantoin should be administered when seizures occur.

0556 REYE'S SYNDROME. (Ger.) Bohles, H. (Universitats-Kinderklinik, 8520 Erlangen, Loschgestr. 15, W. Germany). *Dtsch. Med. Wochenschr.* 102(12):446-451; 1977.

Pathogenetic, clinical, biochemical, and therapeutic aspects of Reye's syndrome (RS) are reviewed. Viruses, toxins (aflatoxin), acetylsalicylic acid, isopropyl alcohol, and short-chained fatty acids have been assumed to be the etiological factors by various authors. It was possible to reproduce the symptoms of RS more or less completely in animals by means of aflatoxins, hypoglycine, and 4-pentaenic acid. RS appears to be the final stage of a pathogenetic process that may be of infectious, toxic, or allergic etiology. The hepatic changes in RS include slight enlargement and high con-

sistency of the liver, diffuse fatty degeneration of the parenchymal cells of hexagonal shape, usually without liver cell necrosis, and mitochondrial degeneration. The fat contains no birefringent lipids. Increased hepatic glycogen was often seen, but cholestasis was never found. The laboratory tests show hypoglycemia and usually a low glucose level in the cerebrospinal fluid, increased glutamic-oxaloacetic and glutamic-pyruvic transaminase activities in the liver, and high blood ammonia levels. None of the therapeutic methods tested or proposed have been substantiated. Peritoneal dialysis, blood exchange, and administration of free blood plasma, glucose with insulin, citrulline, and vitamin K have been used with varying success. Quick diagnosis and intensive treatment are essential. Cooperative studies on RS will be necessary to obtain relevant, reliable findings.

0557 CURRENT STATUS OF THE PROPHYLAXIS OF WILSON'S DISEASE. (Fre.) Sternlieb, I. (Albert Einstein Coll. Medicine, 1300 Morris Park Ave., Bronx, NY 10461). *Rev. Fr. Gastroenterol.* (125):41-44; 1977.

The treatment of 76 Wilson's disease (WD) patients with 1 g/day D-penicillamine is presented. Sixty patients had classical WD with a reduced ceruloplasmin level (CL) and elevated hepatic copper (HC), 3 had a normal CL and elevated HC, 5 had Kayser-Fleischer rings, and 8 had a reduced CL. Two patients who did not receive regular therapy developed psychic symptoms. Side-effects attributed to D-penicillamine were observed only in three patients: elastosis perforans serpiginosa after 16 yr of therapy, nephrosis (the D-penicillamine was replaced by triethylenetetramine), and nephrocalcinosis. No intellectual or physical deterioration, however, occurred in any of the patients during D-penicillamine therapy.

0558 THE PRESENT STATUS OF LIVER TRANSPLANTATION. (Eng.) Calne, R. Y. (Addenbrooke's Hosp., Hills Rd., Cambridge CB2 2QQ, England). *Transplant. Proc.* 9(1):209-216; 1977.

One hundred and fifty-six patients who received liver grafts in Denver (106 patients) and in Cambridge/King's College Hospital series (50 patients) were studied. Of these 106 patients, 27 lived 1 yr or more after liver grafting and 16 were alive at 1-6 yr following grafting. Rejection was the main cause of death in about 10% of the cases. Twenty of the 59 patients age ≤18 yr survived more than 1 yr, and 14 are currently living; one of these is the longest survivor (6.3 yr). Of the 50 patients in the Cambridge/King's series, 12 survived more than 6 months, 4 of these lived beyond 1 yr, and 10 are currently living (one for 2.5 yr). The longest survivor (5.2 yr) in this series died of biliary obstruction secondary to calcified sludge formation blocking the anastomosis between the gallbladder and the recipient common duct. The major complications resulting in death were biliary tract disasters, particularly fistula formation, anastomotic stenosis, and blockage with sludge or superimposed infections, e.g., pneumonia and intraperitoneal abscess or tumor recurrence. A simple method of short-term liver preservation and a new technique of biliary drainage using the gallbladder as a conduit are suggested to produce gratifying results. The low incidence of progressive uncontrollable liver allograft rejection could contribute to an overall understanding of renal and cardiac grafts.

0559 HETEROTOPIC (AUXILIARY) LIVER TRANSPLANTATION IN MAN. (Eng.) Fortner, J. G.; Kim, D. K.; Shiu, M. H.; Yeh, S. D. J.; Howland, W. S.; Beattie, E. J. (Memorial Hosp., 1275 York Ave., New York, NY 10021). *Transplant. Proc.* 9(1):217-221; 1977.

Experience with and results of heterotopic liver transplants (the recipient's liver remains and the donor liver is transplanted to another area) in 7 patients aged 3-72 yr are presented. Six patients had an entire liver transplanted, and one received only a left lobe graft; the latter patient died about 24 hr postoperatively due to acute congestion of the graft, renal failure, and cardiac arrest. Three patients had well-functioning grafts for prolonged periods of time. One is living and well with a normally functioning graft more than 4 yr after transplantation. One patient lived 8 months with a normally functioning graft and died due to infection in her own liver secondary to obstruction from the bile duct cancer. The third patient initially had an uneventful course when a rejection reaction developed; a minimal increase in immunosuppression led to *Pneumocystis carinii* infection and death at 3.5 months. Serial ^{99}Tc scans in one patient showed homogeneous uptake in both the recipient's graft and patient's liver and spleen without apparent significant quantitative difference from 5th to 54th post-transplant day. Rose bengal uptake and excretion were normal. The causes of transplantation failure in six of the cases were multiple abscesses in recipient liver, breakdown of cholecystojejunostomy, hemorrhage and acute congestion of graft, *Pneumocystis* pneumonia, aspiration pneumonia and monilial esophagitis, and septicemia and progressive necrosis. One patient is alive and well. It is suggested that the heterotopic auxiliary liver transplant is clinically useful for selected patients with end-stage noncancerous liver disease.

0560 SELECTIVE CELIAC ANGIOGRAPHY AND SURGICAL EXPLORATION FOR SUSPECTED LIVER CANCER. A COMPARATIVE STUDY OF 196 PATIENTS. (Eng.) Almersjo, O.; Hafstrom, L.; Rosengren, K.; Wedel, N. (Sahlgren Hosp., Univ. Goteborg, Goteborg, Sweden). *Am. J. Surg.* 131(6):676-679; 1976.

0561 PRIMARY HEPATIC CARCINOMA IN THAILAND. (Eng.) Stitnimankarn, T. (Siriraj Hosp.,

Mahidol Univ., Bangkok, Thailand). *Gann Monogr. Cancer Res.* 18:123-127; 1976.

0562 LIVER METASTASES: DIAGNOSTIC VALUE OF BLOOD TESTS, SCINTISCANNING AND LAPAROSCOPY. (Ger.) Angehrn, F. G.; Schmid, P.; Pescia, R.; *et al.* (Stadtspit. Triemli, Zurich. Switzerland). *Dtsch. Med. Wochenschr.* 101(28):1047-1055; 1976.

0563 SEMIQUANTITATIVE ASSESSMENT OF HEPATIC FUNCTION IN OUTPATIENTS BY BREATH ANALYSIS AFTER ORAL ADMINISTRATION OF ^{14}C-AMINOPYRINE. (Eng.) Hanik, L.; Eriksson, S. (Dept. Internal Medicine, Univ. Lund, Lund, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):31; 1977.

0564 THE EFFECT OF HEPATIC DEARTERIALIZATION AND REDEARTERIALIZATION ON CARCINOID LIVER METASTASES. (Eng.) Sivula, A.; Sipponen, P. (Univ. Central Hosp., Helsinki, Finland). *Ann. Chir. Gynaecol.* 65(3):168-175; 1976.

0565 SERUM CHOLESTEROL AND BILE ACID IN PRIMARY HEPATOMA. (Eng.) Hirayama, C.; Irisa, T. (Faculty Medicine, Tottori Univ., Yonago, Japan). *Clin. Chim. Acta* 71(1):21-25; 1976.

0566 SOLITARY SMALL HEPATOCELLULAR CARCINOMA. (Eng.) Mahmood, K.; Schatzki, P. F. (Univ. Pittsburgh Medical Center, Pittsburgh, PA 15213). *Am. J. Dig. Dis.* 21(6):496-502; 1976.

0567 GEOGRAPHIC CORRELATION BETWEEN MORTALITY FROM PRIMARY HEPATIC CARCINOMA AND PREVALENCE OF HEPATITIS B SURFACE ANTIGEN IN GREECE. (Eng.) Trichopoulos, D.; Papaevangelou, G.; Violaki, M.; *et al.* (Univ. Athens Medical Sch., Athens, Greece). *Br. J. Cancer* 34(1):83-87; 1976.

0568 DEMONSTRATION OF A LIVER ABSCESS BY ERC. (Eng.) Weizel, A.; Czygan, P. (Medical Dept., Univ. Heidelberg, Heidelberg, W. Germany). *Endoscopy* 8(2):110-111; 1976.

0569 SURVIVAL AMONG PATIENTS WITH LIVER METASTASES FROM CANCER OF THE COLON AND RECTUM. (Eng.) Fischerman, K.; Peterson, C. F.; Lindkaer Jensen, S.; *et al.* (Rigshospitalet, Copenhagen, Denmark). *Scand. J. Gastroenterol.* 11(Suppl. 37):111-115; 1976.

0570 RESULTS OF LIVER DEARTERIALIZATION COMBINED WITH REGIONAL INFUSION OF 5 FLUOROURACIL FOR LIVER CANCER. (Eng.) Almersjo, O.; Bengmark, S.; Hafstrom, L.; Leissner, K. H. (Dept. Surg. II, Univ. Goteborg, Goteborg, Sweden). *Acta Chir. Scand.* 142(2):131-138; 1976.

0571 HLA AND THE GENETICS OF HEMOCHROMATOSIS [Abstract]. (Eng.) Muir, W. A.; McLaren, G. D.; Braun, W. E.; Schachter, B. Z. (Dept. Medicine, Case Western Reserve Univ., Cleveland, OH). *Clin. Res.* 25(4):573A; 1977.

0572 HISTOCHEMICAL DEMONSTRATION OF LIVER COPPER AND COPPER CARRYING PROTEIN IN CHRONIC LIVER DISEASE [Abstract]. (Eng.) Jain, S.; Scheuer, P. J.; Archer, B.; Newman, S.; Sherlock, S. (Royal Free Hosp., Pond St., London, England). *Clin. Res.* 18(11):A953-A954; 1977.

0573 SERIAL CHANGES IN PLASMA TYROSINE AND PHENYLALANINE LEVELS IN PATIENTS WITH FULMINANT HEPATIC FAILURE TREATED BY POLYACRYLONITRILE HAEMODIALYSIS: RELATIONSHIP TO COMA [Abstract]. (Eng.) Chase, R. A.; Davies, M.; Trewby, P. N.; Silk, D. B. A.; Williams, R. (King's Coll. Hosp. and Medical Sch., London, England). *Gut* 18(11):A952-A953; 1977.

0574 PERIPHERAL MACROPHAGE FUNCTION IN PATIENTS WITH CHRONIC LIVER DISEASE [Abstract]. (Eng.) Triger, D. R.; Kingham, J. G. C.; Lloyd, R. S.; Wright, R. (Dept. Medicine, Southampton Univ., Southampton, England). *Gut* 18(11):A979; 1977.

0575 PROGNOSIS IN CHILDHOOD OF LIVER DISEASE ASSOCIATED WITH ALPHA-1-ANTITRYPSIN DEFICIENCY, TYPE PiZ (ALPHA-1-ANTITRYPSIN DEFICIENCY) [Abstract]. (Eng.) Mowat, A. P.; Psacharopoulos, H.; Williams, R.; Portmann, B.; Tait, R. A. J. (King's Coll. Hosp. Medical Sch., London, England). *Gut* 18(11):A978; 1977.

0576 THE DETECTION OF HETEROZYGOTES IN FAMILIAL INTRAHEPATIC CHOLESTASIS (F.I.C) [Abstract]. (Eng.) Bidot, P.; LaBrecque, D. R.; Hsia, Y. E.; Riely, C. A. (Dept. Human Genetics, Yale Univ., New Haven, CT). *Am. J. Hum. Genet.* 29(6):23A; 1977.

0577 DIAZEPAM KINETICS AND SEDATION IN CHRONIC LIVER DISEASE [Abstract]. (Eng.) McConnell, J. B.; Davis, M.; Curry, S.; Williams, R. (King's Coll. Hosp. and Medical Sch., London, England). *Gut* 18(11):A988; 1977.

0578 PLOIDY PATTERNS OF HUMAN LIVER CELLS IN CHRONIC LIVER DISEASES [Abstract]. (Eng.) Ashihara, T.; Kitamura, O.; Takeoka, O.; Kagawa, K.; Okuda, K.; Takino, T.; Fujita, S. (Shiga Univ. Medical Science, Shiga, Japan). *Gastroenterology* 73(5):1211; 1977.

0579 CULLEN'S SIGN AFTER PERCUTANEOUS LIVER BIOPSY [Letter to Editor]. (Eng.) Capron,

J. P.; Chivrac, D.; Delamarre, J.; Dupas, J. L.; Lorriaux, A. (Centre Hospitalier Universitaire, 80 030 Amiens, France). *Gastroenterology* 73(5): 1185; 1977.

0580 THE DYNAMICS OF AMMONIA METABOLISM IN MAN [Abstract]. (Eng.) Lockwood, A. H.; McDonald, J. M.; Reiman, R. E.; Gelbard, A. S.; Laughlin, J. S.; Duffy, T. E.; Plum, F. (Cornell Medical Coll., Ithaca, NY). *Gastroenterology* 73(5):1232; 1977.

0581 A COMPARATIVE ULTRASTRUCTURAL STUDY OF INHERITED UREA CYCLE ENZYME DEFICIENCY STATES AND REYE'S SYNDROME (RS) [Abstract]. (Eng.) Latham, P.; LaBrecque, D. R.; Kashgarian, M.; Klatskin, G. R. (Dept. Medicine, Yale Univ., New Haven, CT). *Gastroenterology* 73(5):1230; 1977.

0582 LIVER PATHOLOGY IN INHERITED UREA CYCLE ENZYME DEFICIENCY STATES CONTRASTED WITH THAT IN REYE SYNDROME [Abstract]. (Eng.) LaBrecque, D. R.; Latham, P.; Riely, C. A.; Hsia, Y. E.; Klatskin, G. R. (Yale Univ. Sch. Medicine, New Haven, CT). *Gastroenterology* 73(5):1230; 1977.

0583 A METABOLIC EVALUATION OF HEPATIC COMA AND RECOVERY [Abstract]. (Eng.) Hutson, D. G.; Dombro, R.; Zeppa, R.; Ono, J.; Levi, J. U. (Univ. Miami Sch. Medicine, Miami, FL 33152). *Gastroenterology* 73(5):1226; 1977.

0584 CONGENITAL BILIARY FIBROANGIOMATOSIS: ANATOMO-CLINICAL CORRELATION [Abstract]. (Eng.) Patricio, F. R. S.; Fagundes Neto, U.; Wehba, J. (No affiliation given). *Arq. Gastroenterol.* 14(3):161; 1977.

See also, 0004, 0131, 0136, 0184, 0253, 0254, 0255, 0258, 0271, 0283, 0287, 0393, 0400, 0627, 0629, 0635, 0688, 0673, 0681, 0684, 0686, 0709, 0721, 0747, 0748, 0749, 0751, 0752.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

0585 CHOLESTATIC JAUNDICE IN INFANCY: DIAGNOSIS, DIFFERENTIAL DIAGNOSIS AND TREATMENT. (Eng.) Poley, J. R.; Magnani, H. N. (Universitats-Kinderklinik, Prittwitzstrasse 43, 79 Ulm/Donau, W. Germany). *Aust. Paediatr. J.* 12(3): 134-153; 1976.

Common types of persistent cholestatic jaundice (CJ) in infancy are reviewed along with tests for differentiating between intrahepatic cholestasis (IC) and extrahepatic cholestasis (EC). Most infants with CJ may have some type of inflammatory lesion such as seen in neonatal hepatitis (NH) syndromes, or they may have one of the intrahepatic biliary hypoplasia (IBH) syndromes or some type of obstructive lesion of the extrahepatic biliary tree. IBH syndromes probably account for a major group of infants that present with early IC, and patients with IBH present with more severe cholestasis than those with NH. The striking abnormality on biopsy in patients with IBH is the decrease in number and caliber of the bile ducts in portal tracts; sometimes, these bile ducts cannot be identified at all. Three types of lesions characterize EC: true atresia of the common bile duct or hepatic duct, lesions showing obliteration of extensive portions of the sometimes hypoplastic extrahepatic biliary tree, or the absence of recognizable structures reminiscent of bile ducts in the area of the porta hepatis of the hepatoduodenal ligament. The most promising tests for differentiating between IC and EC in infantile CJ are tests for serum 5'-nucleotidase and tests for serum lipoprotein-X (LP-X) combined with cholestyramine (CSM) administration. The absence of serum LP-X in infants with CJ below 7 months of age dictates against a diagnosis of biliary atresia, whereas a positive test (>30 mg/dl) indicates severe cholestasis. When LP-X is measured after CSM administration, a decrease in serum LP-X indicates IC, whereas an increase in serum LP-X indicates EC. Surgical exploration should be reserved for patients in whom an obstructive lesion is unequivocally identified. However, no other test, with the exception of surgery, can determine the type of obstructive lesion present.

0586 STUDY OF THE ETIOLOGY AND PATHOGENESIS OF LOW GRADE NONHEMOLYTIC UNCONJUGATED HYPERBILIRUBINEMIA (GILBERT'S DISEASE). (Eng.) Blueger, A. F.; Krupnikova, E. Z.; Sondore, V. Yu.; Semushina, E. P. (Riga Medical Inst., 12 Padomyu bulv, Riga, USSR). *Acta Hepatogastroenterol. (Stuttg.)* 24(3):140-147; 1977.

Clinical and morphological data on 172 adolescent and adult patients with low-grade nonhemolytic unconjugated hyperbilirubinemia (NUH) were analyzed. The constitutional form (group 1) occurred in 111

patients, the posthepatic form (group 2) in 61. The most common clinical findings were a slight jaundice of the sclerae and abdominal pain plus elevated GOT activity and dysproteinemia. ^{51}Cr RBC survival was shortened in 17/31 patients in group 1 and in 11/15 patients in group 2, the mean values being 16 and 15.5 days, resp., versus a control value of 22.4 days. The mean 24-hr urinary glucuronic acid excretion after menthol ingestion decreased in groups 1 and 2 (1,794 and 1,800 mg, resp.) compared to controls (2,189 mg). Hepatic glucuronyl transferase (GT) activity (77-180 nmol/30 min/mg protein) was low in nine patients and low-normal in one. The mean 24-hr fecal urobilinogen excretion was reduced in groups 1 and 2 (117 ± 35 and 110 ± 45 mg, resp.), but it was increased in 12 patients with hereditary hemolytic jaundice (258 ± 158 mg), compared to control value of 163 ± 15 mg. The hepatocytes showed signs of dystrophy, e.g., vacuolated nuclei, reduced number of microvilli, increased amount of lipofuscin pigment, and large areas of agranular endoplasmic reticulum. Decreased GT activity is an important factor in the pathogenesis of low-grade NUH. The results show not only a disturbance in intrahepatic bilirubin metabolism, but also in other functions of the liver.

0587 PREVENTION OF KERNICTERUS, BASED ON RECENT PROGRESS IN BILIRUBIN CHEMISTRY. (Eng.) Brodersen, R. (Inst. Medical Biochemistry, Univ. Aarhus, DK-8000 Aarhus C, Denmark). *Acta Paediatr. Scand.* 66(5):625-634; 1977.

The binding of bilirubin to albumin, its displacement by fatty acids and drugs, and the importance of plasma pH are reviewed in relation to preventing neurotoxic levels of free bilirubin in kernicterus. Bilirubin IX-α(Z), the only type of bilirubin known to be neurotoxic, is bound reversibly to one high-affinity site on human serum albumin (binding constant at 37 C is 7×10^7 M). Fatty acid levels higher than four times the molar concentration of albumin displace bilirubin from its primary binding site, as do certain sulfonamides and a number of other drugs, thereby increasing the risk of kernicterus. Sulfisoxazole is capable of precipitating kernicterus in icteric infants. Plasma pH also affects the binding of bilirubin to albumin. An infant with a plasma pH of 7.05 (severe acidosis) and a safe upper level of 10 nmol/l of free bilirubin has plasma albumin that is less than half-saturated with bilirubin and a plasma bilirubin concentration of about 150-200 μM. After correction of the pH, the equilibrium is shifted in favor of the plasma albumin, which can now carry a bilirubin concentration of 300-400 μM and still have a tolerable level of the free pigment. For all cases of suspected bilirubin displacement, a rational therapy is the i.v. administration of albumin. More binding sites are then provided for bilirubin as well as for the competitor, and, with exchange transfusion, certain amounts of both bilirubin and competitor are removed from the body.

0588 EFFECT OF INDOOR LIGHTING VS. DARKNESS ON UNCONJUGATED HYPERBILIRUBINEMIA [Abstract]. (Eng.) Zenone, E. A.; Stoll, M. S.; Ostrow, J. D. (Univ. Pennsylvania Medical Service, Philadelphia, PA). *Gastroenterology* 73(5):1256; 1977.

0589 GENETIC INVESTIGATION IN GILBERT'S SYNDROME. (Ger.) Penner, E.; Mayr, W. R.; Djawan, S.; *et al.* (Ordinariat Med. Computerwissensch. Gastroenterol., Univ. Wien, Wien, Austria). *Schweiz. Med. Wochenschr.* 106(25):860-862; 1976.

0590 ALPHA FETOPROTEIN AND NEONATAL JAUNDICE: CONTRIBUTION TO THE STUDY OF PATHOPHYSIOLOGICAL MECHANISM. (Fre.) Tourne, C. E.; Brettes, J. P.; Guez, G.; Ritter, J.; Gandar, R. (C.H.U., B.P. 426/R 5, F 67005 Strasbourg Cedex, France). *J. Gynecol. Obstet. Biol. Reprod. (Paris)* 6(3): 311-317; 1977.

See also, 0153, 0154, 0645.

LIVER AND BILIARY TRACT

Nonviral Hepatitis and Hepatic Injury

0591 THE ROLE OF PLASMA MEMBRANES IN HEPATOTOXIC EFFECTS. (Eng.) Frimmer, M. (Institut fur Pharmakologie und Toxikologie, Fachbereich Veterinarmedizin, Universitat Giessen, D-6300 Giessen, W. Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 297 (Suppl. 1):S15-S19; 1977.

The effect of three different toxins that act directly on hepatic plasma membranes was studied. Phallolysin from *Amanita phalloides* destroyed plasma membranes and killed cells by an outflow of cytoplasm. Leucocidin from *Pseudomonas aeruginosa* produced Ku^+ loss, swelling, and microcirculatory disturbances in perfused livers but the plasma membranes appeared smooth. It is suggested that swelling may be caused by potassium release with simul-

taneous uptake of sodium and water and that the receptor for the pseudomonas toxin might be an integral protein of the plasma membranes. Phalloidin from *A. phalloides* affects the intravascular pressure and the plasma membrane invagination and vacuolization *in vivo*. However, isolated hepatocytes develop typical protrusions in the presence of phalloidin. To understand the mechanism of action of phalloidin, the protective effects of trypsin-like enzymes (proteases) and phospholipase A were studied in isolated hepatocytes. Chymotrypsin and elastase were less potent than trypsin as protectors. The protective activity of phospholipase A depended on the concentration of the enzyme and the time of preincubation. This protection by phospholipase A was reversible within 1 hr after removal of the enzyme by washing. Both protein and lipid structures appear to be involved in the recognition of phalloidin on the surface of liver cells.

0592 PORTAL HYPERTENSION WITHOUT HEPATIC CIRRHOSIS IN CHRONIC VITAMIN A INTOXICATION. (Ger.) Kistler, H. J.; Pluer, S.; Dickenmann, W.; Pirozynski, W. (Medizinische Abteilung, Spital Limmattal, CH-8952 Schlieren, Switzerland). *Schweiz. Med. Wochenschr.* 107(24):825-832; 1977.

Chronic vitamin A intoxication developed in a 37-yr-old man with psoriasis vulgaris following intake of vitamin A (700,000 IU/day, p.o.) for 13 months. Initially, the psoriatic lesions cleared, but skin symptoms returned with further intake of vitamin A. The patient developed symptoms typical of chronic vitamin A intoxication; these included deterioration in his general condition, total alopecia, anorexia, weight loss, ascites, edema of the legs, and depression. The patient had abnormal liver function tests and hepatomegaly. Splenoportography revealed portal hypertension. No signs of liver cirrhosis were demonstrated by repeated liver biopsies. The patient improved gradually after discontinuation of vitamin A. Hair growth was evident, and skin lesions were cured with local therapy. Ascites and edema receded following treatment with spirolactone, ethacrynic acid, and furosemide. The patient was also given a tricyclic antidepressant. Serum vitamin A levels decreased after 3 months, and liver function tests gradually returned to normal; signs of portal hypertension completely disappeared. BSP retention returned to normal after 2 yr.

0593 GLUCOSE-DEPENDENT HEPATIC MEMBRANE TRANSPORT IN NONBACTEREMIC AND BACTEREMIC THERMALLY INJURED PATIENTS. (Eng.) McDougal, W. S.; Wilmore, D. W.; Pruitt, B. A., Jr. (Brooke Army Medical Center, Fort Sam Houston, TX 78234). *J. Surg. Res.* 22(6):697-708; 1977.

Hepatic clearance of indocyanine green (ICG) was measured in 106 studies in 25 thermally injured patients, and the effect of bacteremia and various infused substrates on hepatic dye uptake is described. The patients (mean age, 35 yr) had an average total body surface burn of 59%. Seventeen patients subsequently developed positive blood cultures and were restudied. Fourteen serial measurements of dye concentration taken over a 70-min period were computer fitted to describe each disappearance curve, and the two rate constants, k_1, and k_2, were determined (all $r^2>0.94$). ICG plasma disappearance was unimpaired in nonbacteremic patients when glucose or glucose plus amino acids were part of the parenteral regimen ($-k_1=0.241 \pm 0.023$ and 0.255 ± 0.009/min) but were significantly decreased when near isocaloric amounts of glucose-free amino acid solutions were administered ($-k_1=0.150 \pm 0.016$/min, $p<0.001$). Bacteremic patients had markedly impaired ICG hepatic clearance, irrespective of the hypocaloric dietary regimen. Both exclusion of glucose from the nutrient infusates in nonbacteremic patients and bacteremia resulted in a marked reduction in the maximal velocity of the ICG dye transport reaction. Septic patients infused with glucose and insulin improved their clearance of the dye (from $k_1=0.169 \pm 0.029$ to 0.183 ± 0.028/min, $p<0.01$). These alterations could not be related to changes in circulation or perfusion. Urea production varied inversely and endogenous insulin levels directly with the k_1, suggesting that membrane transport is related to the ornithine cycle and/or is influenced by insulin. Optimal metabolic integrity of the hepatocyte is substrate-specific and dependent upon the provision of exogenous energy. Hepatic transport function is limited in bacteremic patients, as well as in burn patients deprived of glucose. Restoration of hepatic transport processes may be achieved by providing sufficient energy in the form of glucose.

0594 Campylobacter fetus: SEPTICEMIA AND HEPATITIS IN A CHILD WITH AGAMMAGLOBULINEMIA. (Eng.) Wyatt, R. A.; Younoszai, K.; Anuras, S.; Myers, M. G. (Univ. Iowa Hosp., Iowa City, IA 52242). *J. Pediatr.* 91(3):441-442; 1977.

0595 DEXTROPROPOXYPHENE AND JAUNDICE. (Eng.) Ford, M. J.; Kellett, R. J.; Busuttil, A.; Finlayson, N. D. C. (Eastern General Hosp., Edinburgh, Scotland). *Br. Med. J.* 2(6088):674; 1977.

0596 DRUG-INDUCED CHRONIC LIVER DISEASE. (Eng.) Maddrey, W. C.; Boitnott, J. K. (Johns Hopkins Hosp., 601 N. Broadway, Baltimore, MD 21205). *Gastroenterology* 72(6):1348-1353; 1977.

0597 HALOTHANE HEPATITIS: CASE REPORT. (Eng.) Huffman, T. L.; Wilkinson, E. A. (427 Cahill Rd., Branson, MO 64730). *Mo. Med.* 74(6): 261-263; 1977.

Acute Viral Hepatitis

0598 A CRITIQUE OF INTERNATIONAL ASSOCIATION FOR THE STUDY OF THE LIVER (IASL) CRITERIA FOR THE DIAGNOSIS AND TREATMENT OF VIRAL HEPATITIS. (Eng.) Leevy, C. M. (Coll. Medicine and Dentistry of New Jersey, Newark, NJ 07103). *Rush. Presbyterian-St. Lukes Med. Bull.* 15(2):115-122; 1976.

Criteria established by the International Association for the Study of the Liver (IASL) for the diagnosis of viral hepatitis are critically reviewed. A major diagnostic problem relates to the occurrence of hepatitis due to a virus other than the A or B type. This new form of hepatitis, labeled virus C type, is particularly difficult to differentiate from drug-induced hepatitis. Differentiation of alcoholic hepatitis from viral hepatitis has been facilitated by the detection in serum of an antigen to alcoholic hyalin in early phases of the disease followed by the appearance of antibodies to this glycoprotein, which has been isolated and partially purified. Criteria for chronic and fulminant viral hepatitis do not reflect the prognostic implications of these diseases. There is a need to supplement IASL criteria with information on hepatocyte volume, regenerative capacity, and other factors to properly classify patients with fulminant hepatitis. Diagnostic criteria for chronic persistent and chronic active hepatitis are primarily based on histology; serial biopsies and clinical features compatible with either disease may show lesions of the other disease, owing to sampling or a change in status. Clinical tests for evaluating patients with viral hepatitis have been established on three increasingly sophisticated levels, depending on the medical test facilities available. Level I tests include measuring amino transferase activities, serum protein levels, prothrombin time, dye retention, serum bilirubin level and fractionation, and serum alkaline phosphatase activity, plus liver biopsy, and detection of virus B surface antigen and antibody via immunodiffusion, counter immunoelectrophoresis, and inert particle agglutination. Level II tests include measurements of indocyanine green clearance, serum 5' nucleotidase activity, serum bile acid levels, serum immunoglobulin levels, and alpha fetoprotein level, plus hepatitis B surface antigen and antibody detection by complement fixation, radioimmunoassay, and electron microscopy. Level III tests include measurements of galactose elimination capacity, bile acid clearance, lymphocyte response to hepatitis B surface antigen, hepatitis B core and hepatitis B core antibody detection, and the detection of hepatitis A virus by complement fixation, immune adherence test, and fecal viral particles.

0599 THE SIGNIFICANCE OF THE DIAGNOSIS OF ISOENZYMES IN LIVER DISEASES. 2ND COMMUNICATION: ACUTE HEPATITIS AND ITS COURSE. (Ger.) Graubaum, H. J.; Wagenknecht, C.; Klimmt, G.; Baumgarten, R.; Schneider, G.; Schimmelpfennig, W.; *et al.* (Institut Physiologische Biologische Chemie der Humboldt-Universitat, DDR-104 Berlin, Hessische Str. 3/4, E. Germany). *Dtsch. Gesundheitsw.* 32(23): 1087-1092; 1977.

Plasma isoenzyme levels, including glutamate dehydrogenase (GOT), aldolase dehydrogenase (ALD), and LDH were determined in 59 patients with acute hepatitis and correlated with the severity, stage, and prognosis of the disease. Marked increases of total ALD, GOT, and LDH were noted shortly after the appearance of the first prodromal symptoms in the first week. In uncomplicated cases of acute hepatitis, M-type LDH levels returned to normal after 2-3 weeks, liver ALD (ALD-L) and GOT-M after 3 weeks and cytoplasmic GOT (GOT-C) after 5 weeks. H-type LDH increased up to the fifth week. High activities of ALD-L, GOT-C, and M-type LDH and a high level of GOT-M were found to indicate severe cellular damage. Normal activities of ALD-L, GOT-M, and M-type LDH with raised GOT-C demonstrate a normalization of hepatitis (late phase), whereas increases in ALD-L, GOT-C, and M-type LDH above normal indicate the early stage. Elevated enzyme levels beyond the eighth week reflect a protracted course, which may be the start of chronic hepatitis. Following a 2-week course of treatment of one patient with prednisolone (1 mg/kg/day), plasma enzyme levels decreased, whereas the specific activity of the isoenzymes in the liver and plasma, excluding H-type LDH, returned to normal. The decrease of the activities in plasma has repeatedly been attributed to a membrane-stabilizing effect of prednisolone, which does not occur in the liver. Bicycle ergometer tests of five patients with prior hepatitis showed low or constant isoenzyme levels after exertion, whereas all eight controls had elevated isoenzyme levels. These results suggest that isoenzymes are eliminated more rapidly in the former group but more tests are needed to corroborate these findings.

0600 STUDIES OF AN OUTBREAK OF ACUTE HEPATITIS A: I. COMPLEMENT LEVEL FLUCTUATION. (Eng.) Baer, G. M.; Walker, J. A.; Yager, P. A. (P.O. Box 363, Lawrenceville, GA 30246). *J. Med. Virol.* 1(1): 1-7; 1977.

Serum samples from 50 patients who were involved in a hepatitis A outbreak were examined for complement levels and for the presence of antigen-antibody complexes. The outbreak was caused by sewage-contaminated drinking water and involved 49 children and 1 adult. Acute and convalescent serum samples were obtained from the hepatitis patients, and from each of 24 unafflicted matched controls at the same time. Strikingly, the levels of the third and fourth components of complement (C'3 and C'4) were markedly reduced in the acute samples from the sick children

but returned to normal in the convalescent ones; the C'3 and C'4 levels in the healthy matched controls were not reduced. In addition, antigen-antibody complexes were found in convalescent sera from some of the sick children. These findings indicate that immune complexes are present in acute hepatitis A and suggest that the techniques for future virus isolation may have to be altered.

0601 HEPATITIS B: NEW ASPECTS, PRACTICAL CONSEQUENCES, VIROLOGY AND EPIDEMIOLOGY. (Ger.) Deinhardt, F. (Max von Pettenkofer-Institut, Pettenkoferstrasse 9a, D-8000 Munchen 2, W. Germany). *Schweiz. Med. Wochenschr.* 107(15):505-510; 1977.

The epidemiology, virology, serology, and immunization against viral hepatitis are discussed. In addition to the identification and characterization of hepatitis A and B, a third form ("non A-non B" hepatitis) has been recognized. Primary hepatitis is distinct from secondary hepatitis, which frequently is an accompanying symptom of primary infections with Epstein-Barr virus, as seen in infectious mononucleosis. Hepatitis A (short incubation hepatitis) usually occurs as an epidemic and has an incubation time of 20-30 days; it can be prevented or weakened by normal human immune serum globulin (ISG). Hepatitis B (long incubation hepatitis) frequently develops after blood transfusions and is transmitted by blood products and inadequately sterilized instruments or by direct human contact. Perinatal transmission is also possible in children born to mothers who have acute hepatitis B during the last trimester of pregnancy. The incubation time of hepatitis B ranges from 50 to 90 days. Vaccination with a special hepatitis B surface antigen (HBsAg)-ISG (having anti-HBsAg titers of 1:200,000 to 1:500,000 during passive hemagglutination) offers promising protection. Non A-non B hepatitis probably is the most frequent form of post-transfusion hepatitis with an unclear incubation time. There is no cross-immunity between this latter form and the other two. HBsAg is important for the serological diagnosis of hepatitis B; it usually appears in the serum 2-8 weeks prior to the development of clinical symptoms and generally persists for 2-21 weeks. On the other hand, the other components hepatitis B core antigen (HBcAg), e antigen, and virus DNA polymerase can be found temporarily during the time of active virus multiplication. Persistent e antigenemia is found in the severe and active-chronic forms of transient disease and is always accompanied by Dane particles and DNA polymerase, which are indicative of an active process in the liver. Recent trials with vaccines from subunits of HBsAg show promise for controlling hepatitis B by active immunization, but caution is warranted due to the many prevailing difficulties.

0602 MODIFICATIONS OF CHRONIC HEPATITIS-B VIRUS INFECTION IN CHIMPANZEES BY ADMINISTRATION OF AN INTERFERON INDUCER. (Eng.) Purcell, R. H.; London, W. T.; McAuliffe, V. J.; Palmer, A. E.; Kaplan, P. M.; Gerin, J. L.; *et al.* (Natl. Inst. Allergy and Infectious Diseases, Natl. Inst. Health, Bethesda, MD 20014). *Lancet* 2(7989):757-761; 1976.

Chimpanzees chronically infected with hepatitis B virus were treated with the interferon inducer polyriboinosinic-polyribocytidylic acid-poly-l-lysine carboxymethyl cellulose (PICLC). In two separate experiments, the animals received PICLC (3 mg/kg, i.v.) either once daily for 7 days and every other day for an additional week or once daily for two 6-day periods separated by one non-treatment day, and every other day thereafter for 7 weeks. Serum Dane particle-associated DNA polymerase, e antigen, hepatitis B surface antigen, and intrahepatic hepatitis B surface and core antigens diminished during both treatments. Defective (DNA polymerase-negative) Dane particles increased in titer transiently during treatment; these may play a role in the modulation of hepatitis B virus infection. Humoral immune responses in chronic hepatitis B carrier chimps were unaffected. Interferon inducers (or exogenous interferon) may be useful for the treatment of chronic hepatitis B virus infection.

0603 CLINICAL SIGNIFICANCE OF HEPATITIS B ANTIGEN AND OF HEPATITIS B ANTIBODY. (Ger.) Renger, F.; Frank, K. H.; Reimann, W.; Tschopel, L.; Muller, Ch. (Medizinische Klinik, Medizinische Akademie "Carl Gustav Carus," Fetscherstrasse 74, DDR-8019 Dresden, E. Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 36(3/4):121-122; 1976.

The general clinical aspects of hepatitis B antigen (HBAg) and hepatitis B antibody are reviewed. The incidence of carriers of hepatitis B virus is 0.1-20% worldwide. This virus can be transmitted parenterally and orally. HBAg may be present in various body fluids, such as blood, colostrum, sperm, menstrual blood, and feces. The induction of hepatitis is dependent on incubation time, dose inoculated, and the susceptibility of the recipient. The incidence of serum hepatitis can be reduced substantially by weeding out HBAg-positive blood donors and by using blood from one donor instead of pooled blood.

0604 SIMULTANEOUS PRESENCE OF HBsAg AND ANTI-HBs IN THE SERUM OF DIFFERENT SUBTYPES (SEROLOGICAL AND IMMUNOFLUORESCENT STUDIES). (Eng.) Hess, G.; Arnold, W.; Koesters, W.; Biswas, R.; Hutteroth, T. H.; Meyer zum Buschenfelde, K. H. (Freie Universitat Berlin, Klinikum Charlottenburg, 1000 Berlin 19, W. Germany). *Z. Immunitaetsforsch.* 153(2):143-151; 1977.

Three patients with chronic renal insufficiency who were on hemodialysis and receiving blood transfusions were studied both serologically and by direct and indirect immunofluorescence for the presence of various hepatitis B virus antigens and antibodies. Serum factors were measured by immunodif-

fusion, counter-immunoelectrophoresis, passive hemagglutination, and/or radioimmunoassay. Liver biopsy revealed two cases of chronic persistent hepatitis and one case of mild chronic active hepatitis. The two women showed the HBsAg subtype *ad* and anti-HBs with the *y* specificity, while the men carried the HBsAg subtype *ay* and anti-HBs with specificity *d*. No antibody with specificity *a* was found in any serum. Using the Raji-cell technique, no circulating complement binding immune complexes were found. Measurements of rheumatoid factor, antimitochondrial antibodies, smooth muscle antibodies, and antinuclear antibodies were negative, except for the latter in one woman (homogeneous type, 1:10 dilution). All sera were positive for e-antigen and negative for anti-e. One woman was followed for 8 months and persistently carried the HBsAg subtype *ad*; after 4 months she also produced anti-HBs with *y* specificity. Liver biopsy slices tested with subtype-specific antisera showed cytoplasmic fluorescence in all cases with anti-HBs subtypes *ad* and *ay*, while nuclear fluorescence was noted with anti-HBc in two cases. In these two cases positive reaction with anti-human IgG was found in the nuclei but not in the cytoplasm. Indirect immunofluorescence with subtype-specific antisera demonstrated a cytoplasmic fluorescence with anti-HBs subtypes *ad* and *ay* in all patients. Liver sections were positive with HBs antibodies to subtype *d* in two patients (who carried subtype *ad* in their sera) and to the subtype *y* in one patient (who carried *ay* in his serum). Autologous serum produced no fluorescence on liver sections, whereas two patients' sera produced a cytoplasmic reaction on liver slices of the third patient and vice versa. It is suggested that the occurrence of HBsAg and anti-HBs with different subtypes in a single patient may be due to two exposures to hepatitis B viruses of different subtypes, one exposure producing an immune tolerance and another an immune response. Chronic renal insufficiency has been demonstrated to produce an impaired immune competence and might be involved in a differential response to HBsAg subtypes.

0605 CORRELATIONS BETWEEN ANTI-ALBUMIN ANTIBODIES AND HBsAG IN HEPATIC PATIENTS. (Eng.) Lenkei, R.; Babes, V. T.; Dan, M. E.; Mustea, A.; Dobre, I. (Victor Babes Inst., Spl. Independentei 99-1-1, Bucharest 35, 7000 Rumania). *J. Med. Virol.* 1(1):29-34; 1977.

Sera from patients with various hepatic disorders and from healthy controls were examined for the presence of hepatitis B surface antigen (HBsAg) and the incidence and titer of antipolymerized albumin antibodies (AAA). HBsAg detection was done by counterelectrophoresis (CEP) and by reversed passive hemagglutination (RPH). AAA detection was done by immunodiffusion with glutaraldehyde-polymerized human serum albumin (ID-HSA) and by passive hemagglutination (HA-EPA). The incidences of HBsAg in 360 controls, 21 patients with chronic persistent hepatitis, 220 patients with acute viral hepatitis, 64 patients with chronic aggressive hepatitis, and 74 patients with hepatic cirrhosis were 12.8, 52.4, 28.2, 68.8, and 52.7%, resp. The incidences of AAA in these groups were 17.8, 14.3, 69.5, 64.1, and 70.3%, resp., as measured by ID-HSA and 13.9, 33.3, 56.4, 50.0, and 43.2%, resp., as measured by HA-EPA. The AAA concentrations (μg/ml) in these groups were 1,058.3 ± 41.9, 1,075.1 ± 201.2, 479.2 ± 67.6, 504.3 ± 142.4, and 441.3 ± 127.0, resp., as measured by ID-HSA. The AAA titers (log_2) for these groups were 4.3 ± 0.2, 6.5 ± 0.8, 7.0 ± 0.4, 6.4 ± 0.5, and 6.4 ± 0.5, resp., as measured by HA-EPA. The incidence of AAA in ID-HSA did not correlate with HBsAg in any of these groups, but the frequency and titer of HA-EPA were higher in patients with HBsAg in each group, though the difference did not reach statistical significance. There was a significant linear correlation between HBsAg, as measured by RPH, and ID-HSA AAA agglutinins ($r=0.15$, $p<0.001$) and HA-EPA AAA precipitins ($r=0.30$, $p<0.001$). By indirect immunofluorescence, eight sera from patients with acute viral hepatitis and three sera from chronic hepatitis patients (negative in CEP and RPH for HBsAg and with high titers in HA-EPA for AAA) were tested for anti-hepatitis B core antibodies (anti-HBc). In eight cases, a bright fluorescence was obtained in the same nuclei as those revealed by standard positive sera. No fluorescence was obtained by the direct application of sheep anti-human immunoglobulin serum labeled with fluorescein isothiocyanate or by other sera from normal subjects previously tested for HBsAg by RPH and for anti-HBc by indirect immunofluorescence. The results indicate that indirect immunofluorescence for the detection of anti-HBc is more sensitive than RPH in the diagnosis of a recent or ongoing hepatitis B virus infection.

0606 HBsAg and HBcAg IN THE LIVERS OF ASYMPTOMATIC HEPATITIS B ANTIGEN CARRIERS. (Eng.) Tapp, E.; Jones, D. M. (Preston Royal Infirmary, Preston, England). *J. Clin. Pathol.* 30(7):671-677; 1977.

Liver sections from 33 asymptomatic blood donors (22 of whom had multiple biopsies) with constant hepatitis B surface antigen (HBsAg) antigenemia were studied by immunoperoxidase and orcein staining and by electron microscopy. With immunoperoxidase, HBsAg was noted in the liver cell cytoplasm of 34/48 biopsies; 12/33 carriers were negative. Staining varied from a small part of the cell to the entire cell with an intensity from pale yellow to dark brown. A foamy cytoplasm indicated unstained lipid droplets. There was no indication HBsAg was confined to cellular margins. Sheets of cells with diffuse staining affecting up to two-thirds of the lobule were seen, while another group of cells could be discerned in which considerably fewer cells were stained. The diffuse pattern was frequent in livers with focal parenchymal necrosis and was seen in two-thirds of biopsies showing cirrhosis. Focal staining was commonly found in cases of chronic aggressive hepatitis. Chronic persistent hepatitis showed both patterns of staining with equal frequency. Subsequent biopsies indicated no change in pattern, with about half the carriers in each group showing some histological deterioration; however, only 1/10

follow-up biopsies showed deterioration in carriers negative for HBsAg. Only 8 (from 4 donors) of 48 biopsies were positive for hepatitis B core antigen (HBcAg) in hepatocyte nuclei, with immunoperoxidase staining varying from a brown to black color. Orcein, reactive for HBsAg, stained only about half the biopsies positive with immunoperoxidase. With electron microscopy, filaments and tubules were observed within dilated endoplasmic reticulum. In 6 (from 3 donors) of 37 biopsies, 20-nm particles were found in the cell nuclei, and those biopsies were all positive with the HBcAg immunoperoxidase procedure. The results indicate that the immunoperoxidase technique is more sensitive than the orcein procedure, that there is an inverse relationship between the content of HBsAg in the cytoplasm and the degree of liver cell destruction, and that HBcAg occurs only in the liver cell nuclei of individuals with chronic hepatitis of the aggressive or persistent type.

0607 e-SYSTEM AND INTRAHEPATOCELLULAR HBcAG AND HBsAG IN HBsAG POSITIVE PATIENTS WITH LIVER DISEASES AND HEALTHY CARRIERS. (Eng.) Hess, G.; Nielsen, J. O.; Arnold, W.; Meyer zum Buschenfelde, K. H. (Second Dept. Internal Medicine, Univ. Mainz, Mainz, W. Germany). *Scand. J. Gastroenterol.* 12(3): 325-330; 1977.

To investigate the prognostic value of the e antigen and the importance of the e antigen as a sign of infectivity, patients with hepatitis B surface antigen (HBsAg)-positive liver diseases and healthy carriers were studied for the presence of e antigen and antibody (anti-e) as well as for intrahepatocellular HBsAg and hepatitis B core antigen (HBcAg). The e antigen was demonstrated in 9 of 12 patients with chronic persistent hepatitis, in 15 of 39 patients with chronic active hepatitis, in 3 of 40 patients with acute type B hepatitis, and in 2 of 9 patients with a protracted course of type B hepatitis. No e antigen was found in healthy HBsAg carriers in patients with complete recovery from type B hepatitis 1 yr after the onset of the disease. Anti-e was detected in 24 of 61 healthy HBsAg carriers with a normal liver histology and in one patient with a mild form of a chronic persistent hepatitis. The presence of e antigen in serum was associated with the presence of HBcAg in the nuclei of the liver cells. Twenty-seven of 29 e-positive patients had HBcAg in the liver cell nuclei. In contrast, none of 20 patients with anti-e in the serum had HBcAg in the liver cells. The results support the hypothesis that the sera of healthy HBsAg carriers are not infectious.

0608 DISCORDANT e ANTIGEN, DNA POLYMERASE ACTIVITY, AND DANE PARTICLE RESPONSES IN TWO PATIENTS REPRESENTING AN INDEX CASE-CONTACT CASE PAIR WITH HEPATITIS B VIRUS INFECTION. (Eng.) Perrillo, R. P.; Gelb, L. D.; Milligan, W. H., III; Wellinghoff, W.; Aach, R. D. (Washington Univ. Sch. Medicine, St. Louis, MO 63110). *J. Infect. Dis.* 136(1):117-121; 1977.

Sera from an index case-contact case pair with hepatitis B virus infection were examined for the presence of e antigen and antibody to e antigen (anti-e), as well as for DNA polymerase activity and the frequency of Dane particles. The index case was a 30-yr-old physician who had no known history of exposure, but who developed acute viral hepatitis. Six months later, chronic aggressive hepatitis was noted. Two months after the onset of illness in the first patient, his wife, who had no known contact other than her husband, developed anicteric, acute viral hepatitis. Six months later, biopsy revealed chronic aggressive hepatitis. Sera from the index patient were consistently e-antigen negative during the 12-month study period, while sera from the contact patient were e-antigen positive during the same period. The contact patient's sera demonstrated an e antigen/anti-e precipitin line at a serum concentration of 1:16 while sera from the index patient failed to manifest this line, even at a threefold concentration of serum. Sera from both patients were positive for hepatitis B surface antigen (HBsAg) during the entire test period, and neither patient had detectable anti-e or antibody to HBsAg. Sera from three healthy persons and from two HBsAg-negative patients were used as controls for the detection of Dane particles and DNA polymerase activity. None of the controls demonstrated any Dane particles, while the index case had between one and five particles per field, and his wife had over 16 particles per field. The DNA polymerase activity of the index patient did not differ from that of controls, while that of the contact patient was unusually high (2,570 counts/min). Sera of both patients appeared to be of the HBsAg subtype *adw*. These responses demonstrate that there is a lack of correlation between the presence of e antigen and the viral strain, and they strengthen the relationship of the e antigen to persistent viremia. The role of the host, rather than that of the infective agent, in the expression of the hepatitis B antigen is emphasized.

0609 HOST RESPONSES TO HEPATITIS B INFECTION IN PATIENTS IN A CHRONIC HEMODIALYSIS UNIT. (Eng.) London, W. T.; Drew, J. S.; Lustbader, E. D.; Werner, B. G.; Blumberg, B. S. (Inst. Cancer Res., 7701 Burholme Ave., Philadelphia, PA 19111). *Kidney Int.* 12(1):51-58; 1977.

To study the nature and variation of hepatitis B infection in patients undergoing chronic hemodialysis, host responses to hepatitis B infection were studied in 222 patients in a chronic hemodialysis unit. From 1970 to 1976, patients were monitored monthly for development of hepatitis B surface antigen (HBsAg), antibody to HBsAg (anti-HBs), and SGPT elevations. Five categories of patients were identified: (1) chronic carriers of HBsAg, (2) transiently HBsAg(+), who developed anti-HBs, (3) HBsAg(-) on admission, who developed anti-HBs without becoming HBsAg(+), (4) anti-HBs(+) on admission, and (5) uninfected, who remained HBsAg(-) and anti-HBs(-). For patients who became HBsAg(+), the probability of becoming a chronic carrier was

62.8% and rose to 88.5% if they had been HBsAg(+) for 5 consecutive months. Men were more likely to become chronic carriers, and women were more likely to develop anti-HBs. Neither age, race, nor type of underlying kidney disease was associated with particular host responses to hepatitis B virus. No effect of hepatitis B infection on mortality was detected. Variation in host response to hepatitis B infection among renal dialysis patients may affect the usefulness of hepatitis B hyperimmune globulin and hepatitis B vaccine and may be related to the outcome of kidney transplantation.

0610 THE EFFECT OF INTRADERMAL ADMINISTRATION OF Corneybacterium parvum ON THE IMMUNE RESPONSE TO HEPATITIS Bs ANTIGEN. (Eng.) Papaevangelou, G.; Sparros, L.; Vissoulis, C.; Kyriakidou, A.; Giokas, G.; Hadzimanolis, J.; *et al.* (Univ. Athens, Medical Sch., Athens 609, Greece). *J. Med. Virol.* 1(1):15-19; 1977.

Because the persistence of hepatitis B surface antigen (HBsAg) in asymptomatic chronic carriers for years or for life has been attributed to a specific defect in the immune response to HBsAg, the immunopotentiating effect of the intradermal administratio of a course of four doses (0.25 ml) of a standard suspension of killed *Corynebacterium parvum* (2 mg/ml) was studied in 10 asymptomatic chronic HBsAg carriers, in 11 persons with antibodies to HBsAg (anti-HBs), and in 6 persons without HBsAg or anti-HBs. HBsAg, anti-HBs, and leukocyte migration inhibition (LMI) studies were performed in pre- and postinoculation blood samples. *C. parvum* produced a substantial increase of anti-HBs titer in subjects with preexisting anti-HBs. However, anti-HBs responses were not induced in carriers. HBsAg was not eliminated, and its titer remained practically unchanged in carriers. These results support the hypothesis that in carriers the specific defect in the immune response to HBsAg possibly exists at the B cell level.

0611 NECROSIS OF THE HEPATOCYTES WITH HEPATITI B SURFACE ANTIGEN: OCCURRENCE IN A CHRON HEPATITIS B SURFACE ANTIGEN CARRIER. (Eng.) Karasawa, T.; Shikata, T. (Faculty Medicine, Univ. Toky Tokyo, Japan) *Arch. Pathol. Lab. Med.* 101(6):280-285; 1977.

Light and electron microscopy of a liver specimen from a 25-yr-old man who was an asymptomatic chronic hepatitis B surface antigen (HBsAg) carrier for at least 1 yr and 8 months are reported. No anti-HBsAg was demonstrated in the serum of this patient. The microscopic findings revealed small lymphocytes and macrophages in close contact with liver cells, partial hepatocyte cytolysis of varying degrees, lytic hepatocyte necrosis, and the complete loss of a few hepatocytes with HBsAg in the cytoplasm. HBsAg or HBsAg-associated material in the cytoplasm was found only in either necrotic liver cells or in liver cells with ground glass appearing cytoplasm. Conventional immunofluorescence studies revealed HBsAg not only in the cytoplasm of hepatocytes but also on the cell membrane. Only hepatocytes with HBsAg both in the cytoplasm and on the cell membrane were necrotic. No IgG or IgM was detected on the cell membrane of the liver cells or in the necrotic foci. Complement component 3 was not present in the necrotic foci. The above evidence most likely rules out not only the involvement of cytotoxic antibody- and complex-mediated humoral immune responses but also that of antibody-directed cell-mediated cytotoxicity or of a cytotoxic mechanism whereby nonspecific normal lymphoid cells (supposedly either bone marrow-derived lymphocytes or macrophages) destroy target cells coated with antibody molecules. Thus, the liver cell injury in this case was probably due to specific contact of hepatocytes with killer cells which are immunologically activated thymus-derived lymphocytes (contact killing).

0612 PERIPHERAL BLOOD AND LIVER TISSUE T AND B LYMPHOCYTES AND HEPATITIS B VIRUS (HBV) [Letter to Editor]. (Eng.) Miller, D. J.; Dwyer, J. M.; Klatskin, G. (Yale Univ. Sch. Medicine, New Haven, CT 06510). *Gastroenterology* 74(1):160-161; 1978.

0613 A CLINICOPATHOLOGIC STUDY OF FULMINANT HEPATITIS [Abstract]. (Eng.) Arakawa, Y.; Fujita, S. (Nihon Univ. Sch. Medicine, Tokyo, Japan). *Gastroenterol. Jpn.* 12(4):326; 1977.

0614 COMPLEMENT IN FULMINANT HEPATITIS [Abstract]. (Eng.) Kondo, M.; Torisu, M. (Kyoto Prefectural Univ. Medicine, Kyoto, Japan). *Gastroenterol. Jpn.* 12(4):329; 1977.

0615 PLASMA EXCHANGE USING CELLTRIFUGE IN PATIENT WITH FULMINANT HEPATITIS [Abstract]. (Eng.) Iwanaga, T.; Horiuchi, A. (Kinki Univ. Sch. Medicine, Osaka, Japan). *Gastroenterol. Jpn.* 12(4):329; 1977.

0616 THE MEASUREMENT OF SERUM PROTEINS WITH RAPID TURNOVER FOR EARLY DIAGNOSIS OF FATAL HEPATITIS [Abstract]. (Eng.) Kobayashi, K.; Kameda, S.; Hattori, N. (Sch. Medicine, Kanazawa Univ., Kanazawa, Japan). *Gastroenterol. Jpn.* 12(4):328; 1977.

0617 CHARCOAL HEMOPERFUSION FOR THE TREATMENT OF FULMINANT HEPATITIS [Abstract]. (Eng.) Amano, I.; Kano, H. (Chukyo Hosp., Nagoya, Japan). *Gastroenterol. Jpn.* 12(4):327; 1977.

0618 TREATMENT OF FULMINANT HEPATITIS BY NEW ARTIFICIAL LIVER SUPPORT SYSTEM (PLASMA PERFUSION DETOXIFICATION) [Abstract]. (Eng.) Inoue, N.; Sakai, T. (Oji Natl. Hosp., Tokyo, Japan). *Gastroenterol. Jpn.* 12(4):328; 1977.

0619 BACKGROUND AND PRACTICAL ASPECTS OF HEPATITIS B SURFACE ANTIGEN CARRIER STATE. (Eng.) Helske, T. (Finnish Red Cross Blood Transfusion Service, Helsinki, Finland). *Ann. Clin. Res.* 8(3):158-161; 1976.

0620 DETECTION OF HEPATITIS B ANTIGEN IN BLOOD DONORS BY RADIOIMMUNOASSAY AND 3 REVERSE PASSIVE HAEMAGGLUTINATION TECHNIQUES. (Eng.) Seidl, S.; Ziegler, G. B. (Inst. Immunohematology, Univ. Frankfurt/M., Frankfurt, W. Germany). *Vox. Sang.* 31(2):81-86; 1976.

0621 THE PREVENTION OF VIRAL HEPATITIS. (Eng.) Krugman, S. (New York Univ. Medical Center, New York, NY). *Ann. Clin. Res.* 8(3):216-220; 1976.

0622 THE LONG TERM OUTCOME OF HEPATITIS B. (Eng.) Norkrans, G.; Hermodsson, S.; Lundin, P.; Iwarson, S. (Dept. Infectious Diseases, Univ. Goteborg, Goteborg, Sweden). *Infection* 4(2): 70-72; 1976.

0623 HEPATITIS B ANTIGEN AND SYSTEMIC LUPUS ERYTHEMATOSUS. FALSE POSITIVE COMPLEMENT FIXATION DUE TO ANTI ANTIBODIES. (Eng.) Celada, A.; Nydegger, U. E.; Lambert, P. H.; Miescher, P. A. (Hop. Cant., Geneva, Switzerland). *Klin. Wochenschr.* 54(7):303-307; 1976.

0624 STUDY OF INTEROBSERVER VARIATION BETWEEN LIVER HISTOPATHOLOGISTS [Abstract]. (Eng.) Theodossi, A.; Knill-Jones, R. P.; Silk, D. B. A.; Williams, R. (King's Coll. Hosp., Denmark Hill, London, England). *Gut* 18(11):A953; 1977.

0625 EPIDEMIOLOGICAL STUDY OF NEONATAL HEPATITIS SYNDROME IN INFANCY: INCIDENCE AND EARLY COURSE [Abstract]. (Eng.) Mowat, A. P.; Psacharopoulos, H.; Williams, R. (King's Coll. Hosp. Medical Sch., London, England). *Gut* 18(11):A977; 1977.

0626 MOTHER-TO-INFANT TRANSMISSION OF HEPATITIS B VIRUS [Abstract]. (Eng.) Tarlow, M. J.; Derso, A.; Boxall, E. H.; Flewett, T. H. (East Birmingham Hosp., Birmingham, England). *Gut* 18(11): A964; 1977.

0627 LEVAMISOLE IN THE TREATMENT OF HBs ANTIGEN POSITIVE CHRONIC ACTIVE LIVER DISEASE [Abstract]. (Eng.) Chadwick, R. G.; Jain, S.; Thomas, H. C.; Sherlock, S. (Royal Free Hosp., London, England). *Gut* 18(11):A979; 1977.

0628 HEPATITIS B: ABSENCE OF TRANSMISSION BY GASTROINTESTINAL ENDOSCOPY [Abstract]. (Eng.) McClelland, D. B. L.; Burrell, C. J.; Tonkin, R. W.; Heading, R. C. (Dept. Therapeutics, Univ. Edinburgh, Edinburgh, Scotland). *Gut* 18(11): A968; 1977.

0629 MICROSOMAL DRUG OXIDASES IN LIVER DISEASE AND AFTER CHRONIC DRUG INGESTION IN MAN [Abstract]. (Eng.) Farrell, G. C.; Cooksley, W. G. E.; Powell, L. W. (Dept. Medicine, Univ. Queensland, Brisbane, Australia). *Gastroenterology* 73(5):1219; 1977.

0630 HEPATITIS B 'e' ANTIGEN: AN ASSOCIATION WITH LACTATE DEHYDROGENASE ISOZYME-5 [Abstract]. (Eng.) Vyas, G. N.; Peterson, D. L.; Townsend, R. M.; Damle, S. R.; Magnius, L. O. (Dept. Lab. Medicine, Univ. California, San Francisco, CA). *Gastroenterology* 73(5):1253; 1977.

0631 INTRAHEPATIC LOCALIZATION OF THE SURFACE (HBsAg) AND CORE (HBcAg) ANTIGENIC DETERMINANTS ASSOCIATED WITH HEPATITIS B VIRUS IN BIOPSY SAMPLES FROM PATIENTS WITH LIVER DISEASE. (Eng.) Rizzetto, M.; Crivelli, O.; Suriani, R.; Verme, G. (Osp. Mauriziano Umberto I, Torino, Italy). *Ric. Clin. Lab.* 6(1):41-54; 1976.

0632 HEPATITIS B VIRUS IN NUCLEOLI OF LIVER CELLS [Letter to Editor]. (Eng.) Michalak, T.; Nowoslawski, A. (Natl. Inst. Hygiene, 00-791 Warsaw, Poland). *N. Engl. J. Med.* 297(14): 787-788; 1977.

0633 TYPE A VIRAL HEPATITIS AND ITS IMPORTANCE IN HYGIENIC MEASURES IN THE SURROUNDING ENVIRONMENT. (Rum.) Romanescu, Gh. (Laboratorul de epidemiologie al Sectiei pentru controlul de stat al serurilor si vaccinurilor, Bucharest, Rumania). *Rev. Ig. [Bacteriol.]* 26(2):109-112; 1977.

0634 INITIAL EXPERIENCE WITH THE APPLICATION OF THE HEMOSORPTION METHOD IN THE TREATMENT OF CHILDREN WITH HEPATIC INSUFFICIENCY. (Rus.) Burkov, I. V.; Osipov, Yu. N.; Mosharov, O. P.; Uchaikin, V. F.; Troitsky, A. A. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Vestn. Akad. Med. Nauk SSSR* (6):52-57; 1977.

See also, 0538, 0551, 0567, 0575, 0637, 0638, 0673.

Chronic Hepatitis

0635 LIVER BIOPSY IN HEMOPHILIA A. (Eng.) Leseane, H. R.; Morgan, J. E.; Blatt, P. M.; Webster, W. P.; Roberts, H. R. (Univ. North Carolina Sch. Medicine, Chapel Hill, NC 27514). *Ann. Intern. Med.* 86(6):703-707; 1977.

The results of percutaneous liver biopsy, under the coverage of glycine-precipitated factor VIII, in six patients with hemophilia A who had persistently abnormal liver function tests for at least 6 months, are described. Each patient was checked for the presence of an inhibitor of factor VIII; if no inhibitor was present, the patient received an infusion of glycine-precipitated factor VIII calculated to raise his factor VIII level to 100%. Blood was drawn after the infusion, and a factor VIII assay was done to be certain that the *in vivo* recovery and half-life of factor VIII were normal. If inhibitor was found, the biopsy was not done. Factor VIII, calculated to raise the factor VIII level to 100%, was administered just before the biopsy. The level was kept at about 75% of normal for 72 hr after the biopsy. Three patients had chronic active hepatitis, and three had chronic persistent hepatitis. No complications were encountered as a result of the biopsy procedure. These results suggest that percutaneous liver biopsy should be considered in patients with hemophilia A with continuously abnormal liver function tests to establish a histologic diagnosis and to guide further therapy.

0636 FOLLOW-UP OBSERVATION OF 52 PATIENTS WITH CHRONIC AGGRESSIVE HEPATITIS. (Ger.) Wyss, G.; Stalder, G. A.; Bianchi, L. (Medizinische Universitats-Poliklinik, Hebelstrasse 1, CH-4056 Basel, Switzerland). *Schweiz. Med. Wochenschr.* 107 (15):520-523; 1977.

The results of the regular clinical, biochemical, and bioptic follow-up of 52 patients (31 men, 21 women, average age 49.5 yr) with histologically proven chronic aggressive hepatitis (CAH) are presented. The length of the follow-up was 0.5-9 yr (average 31 months). The pathological findings included hepatomegaly in 15 cases, spider nevi in 9, palmar erythema in 7, Dupuytren's contracture in 3, and ascites in 3. Pathological serum bilirubin levels were found in 19%, pathological serum glutamic-oxaloacetic transaminase levels in 89%, pathological serum glutamic-pyruvic transaminase levels in 91%, and pathological alkaline phosphatase levels in 13.5%. Fourteen men and 9 women were HBsAg-positive; 4 men and 3 women (among 42 patients) were HBs-antibody-positive; antinuclear antibodies were found in 16 men and 13 women, and anti-smooth muscle antibodies in 17 men and 13 women. The first biopsy revealed CAH in 64% of all cases. Twenty-two patients already had liver cirrhosis at the time of the histological diagnosis of CAH. Six patients were treated with the combination of imurel and prednisone on the basis of adequate indications (histological diagnosis, chronic process, tenfold increase of the transaminase level or fivefold increase of the transaminase level with twofold increase of the gamma-globulin level). Five patients responded to the therapy. Six patients died during the follow-up; the cause of death was hemorrhage of esophageal varices in one case, hepatoma in two cases, bronchial carcinoma in two cases, and carcinoma of the pancreas in one case. The clinical and laboratory findings remained fairly constant during the follow-up period in patients with mild CAH. Exacerbation was seen only in those patients in whom the first biopsy revealed decompensated liver cirrhosis and ascites.

0637 SEROLOGICAL EVIDENCE OF HEPATITIS B INFECTION IN PATIENTS WITH CHRONIC LIVER DISEASES: RADIOIMMUNOASSAY OF HBsAg AND ANTI-HBs. (Eng.) Skinhoj, P.; Nielsen, J. O.; Dietrichson, O. (Dept. Infectious Diseases, Rigshospitalet, Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(5):615-619; 1977.

The prevalence of hepatitis B surface antigen (HBsAg) and anti-HBs was determined by a sensitive double-antibody radioimmunoassay technique in 394 patients with chronic liver diseases. The patients were divided into five groups on the basis of information about their alcohol consumption and liver diseases, i.e., patients with 1) cirrhosis of the liver (199), 2) primary biliary cirrhosis (18), 3) steatosis (79), 4) chronic active hepatitis without cirrhosis (74), and 5) chronic persistent hepatitis (24). HBsAg was demonstrated in 37 of 98 consecutive patients with chronic hepatitis and in 14 of 108 patients with nonalcoholic cirrhosis. HBsAg was not found in any of the patients with steatosis, alcoholic cirrhosis, or primary biliary cirrhosis. In the patients with chronic hepatitis, HBsAg or anti-HBs was demonstrated in 83% of the patients below 40 yr of age in contrast to 48% of the patients over 40 yr of age. It is concluded that, in Denmark, the majority of patients with chronic aggressive or persistent hepatitis have serological evidence of previous contact with hepatitis B virus (HBV) infection. This is in contrast to patients with other chronic liver diseases, in whom only a minor proportion of the nonalcoholic cases appear to have been exposed to HBV infection.

0638 DNA BINDING ANTIBODIES IN HBsAg POSITIVE AND NEGATIVE ACUTE AND CHRONIC LIVER DISEASE [Abstract]. (Eng.) Kingham, J. G. C.; Ganguly, N. K.; Holgate, S. T.; Triger, D. R.; McGuire, M.; Cohen, B.; *et al.* (Southampton

General Hosp., Southampton, England). *Gut* 18(11): A978-A979; 1977.

0639 RELATIVE VALUE OF LYMPHOCYTE CYTOTOXICITY FOR HEPATOCYTES AND CONVENTIONAL BLOOD TESTS IN ASSESSMENT OF HISTOLOGICAL ACTIVITY IN CHRONIC ACTIVE HEPATITIS [Abstract]. (Eng.) Cochrane, A. M. G.; Portmann, B.; Eddleston, A. L. W. F.; Williams, R. (King's Coll. Hosp. and Medical Sch., London, England). *Gut* 18(11):A978; 1977.

0640 TREATMENT OF HBsAg-POSITIVE CHRONIC ACTIVE HEPATITIS WITH HUMAN FIBROBLAST INTERFERON [Abstract]. (Eng.) Kingham, J. G. C.; Ganguly, N. K.; Shaari, Z. D.; Holgate, S. T.; McGuire, M. J.; Mendelson, R.; *et al.* (Southampton General Hosp., Southampton, England). *Gut* 18(11):A952; 1977.

0641 CONTROLLED TRIAL OF CYCLOPHOSPHAMIDE IN ACTIVE CHRONIC HEPATITIS [Abstract]. (Eng.) Gilmore, I. T.; Cowan, R. E.; Axon, A. T. R.; Thompson, R. P. H. (St. Thomas' Hosp., London, England). *Gut* 18(11):A952; 1977.

0642 CHRONIC LOBULAR HEPATITIS: A DISTINCT ENTITY [Abstract]? (Eng.) Wilkinson, S. P.; Portmann, B.; Cochrane, A. M. G.; Williams, R. (King's Coll. Hosp. and Medical Sch., London, England). *Gut* 18(11):A951-A952; 1977.

0643 NATURAL HISTORY OF CHRONIC PERSISTENT HEPATITIS [Abstract]. (Eng.) Chadwick, R. G.; Galizzi, J.; Heathcote, E. J.; Cohen, B.; Scheuer, P. J.; Sherlock, S. (Royal Free Hosp., London, England). *Gut* 18(11):A951; 1977.

0644 RADIOIMMUNOASSAY FOR ANTIBODIES TO HUMAN LIVER-SPECIFIC MEMBRANE LIPOPROTEIN (LSP) IN CHRONIC ACTIVE HEPATITIS AND CHRONIC PERSISTENT HEPATITIS [Abstract]. (Eng.) Jensen, D.; McFarlane, I. G.; Eddleston, A. L. W. F.; Williams, R. (King's Coll. Hosp. and Medical Sch., London, England). *Gut* 18(11):A978; 1977.

0645 NON-FAMILIAL UNCONJUGATED HYPERBILIRUBINEMIA AND REDUCED HEPATIC BILIRUBIN UDP-GLUCURONYL TRANSFERASE ACTIVITY IN PATIENTS WITH CHRONIC PERSISTENT HEPATITIS [Abstract]. (Eng.) Felsher, B. F.; Carpio, N. M. (Long Beach Veterans Admin. Hosp., Long Beach, CA). *Gastroenterology* 73(5):1220; 1977.

See also, 0538, 0563, 0572, 0577, 0598, 0607, 0685.

LIVER AND BILIARY TRACT

Alcoholic Liver Disease

0646 PHARMOKINETICS AND PHARMACODYNAMICS OF ETHANOL, WHISKEY, AND ETHANOL WITH n-PROPYL, n-BUTYL, AND ISO-AMYL ALCOHOLS. (Eng.) Auty, R. M.; Branch, R. A. (Fisons Ltd., Pharmaceutical Div., Res. and Development Lab., Bakewell Rd., Loughborough Leics, England). *Clin. Pharmacol. Ther.* 22(2):242-249; 1977.

To determine whether higher aliphatic alcohols exert any effect on the pharmacology of ethanol, plasma ethanol concentration, reaction time, and EEG were recorded in six normal men after ingestion of ethanol alone (group 1), whiskey (group 2), or a mixture of ethanol, n-proponol, n-butanol, and iso-amyl alcohol (group 3). The peak plasma ethanol concentration and the total area under the plasma concentration:time curve of ethanol did not depend upon the type of drink given, but the half-life of the terminal exponential phase of ethanol elimination was longer in group 3. In each study period, reaction time increased, there was a relative increase in delta activity (2-3 Hz), and a fall in mean dominant frequency in EEG activity. The extent of increase in reaction time depended on the rate of increase in plasma concentration and correlated with the concentration of ethanol while the plasma concentration of ethanol was falling. Differences in the effects of ethanol between study periods were minimal.

0647 ULTRASTRUCTURE OF PERIPORTAL AND CENTRILOBULAR HEPATOCYTES IN HUMAN FATTY LIVER OF VARIOUS AETIOLOGY. (Eng.) Petersen, P. (Rigshospitalet, 2100 Copenhagen, Denmark). *Acta Pathol. Microbiol. Scand.* [*A*] 85(3):421-427; 1977.

The ultrastructure of periportal and centrilobular hepatocytes of two alcoholic patients with fatty liver, two diabetic patients, and two obese patients were compared with that of hepatocytes in two patients with normal livers. A striking difference between periportal and centrilobular hepatocytes

in all cases of fatty liver was demonstrated. The periportal hepatocytes contained abnormal mitochondria, while centrilobular hepatocytes had normal mitochondria, but fatty vacuoles, peroxisomes, and lysosomes were more abundant. The nature and degree of ultrastructural changes showed no correlation with etiology of the fatty liver. The normal livers showed no such changes or distinct differences between periportal and centrilobular hepatocytes.

0648 THE HEPATIC CONVERSION OF VITAMIN D IN ALCOHOLICS WITH VARYING DEGREES OF LIVER AFFECTION. (Eng.) Lund, B.; Sorensen, O. H.; Hilden, M.; Lund, B. (Frederiksberg Hosp., Copenhagen, Denmark). *Acta Med. Scand.* 202(3):221-224; 1977.

The seasonal variations in circulating 25-hydroxycholecalciferol (25-HCC) were studied in 102 alcoholics with fatty liver disease without histologic signs of cirrhosis, in 35 patients with alcoholic cirrhosis, and 596 normal controls. Alcoholics had generally lower 25-HCC values than did the controls, particularly in the summer. This was explained primarily by insufficient diet and reduced exposure to sunshine. The ability of the liver to hydroxylate in the 25-position was studied in three groups of alcoholics with fatty liver disease without cirrhosis, compensated cirrhosis, or severely incompensated cirrhosis. All three groups exhibited a significant increase in serum 25-HCC following the p.o. administration of cholecalciferol (1,200 U daily for 7 days). Similar rises were seen 7 days after a single injection of 10,000 U cholecalciferol. These findings indicate a normal intestinal absorption of vitamin D, even in advanced alcoholic liver disease, and are inconsistent with a severely damaged 25-hydroxylation capacity in these patients. Osteomalacia due to impaired liver hydroxylation of vitamin D cannot therefore explain the increased fracture rate and the decreased bone mass that have been described in alcoholics.

0649 COMPARISON OF FEATURES OF ALCOHOLIC LIVER DISEASE [Abstract]. (Eng.) Connell, A. M.; Weir, D.; Fitzgerald, O.; McDonald, W.; Whelton, M.; McCarthy, C.; *et al.* (Univ. Hosp., Cincinnati, OH). *Gut* 18(11):A968; 1977.

0650 CAN WE BELIEVE THE FIGURES? DEATHS FROM CIRRHOSIS AND HOSPITAL PREVALENCE [Abstract]. (Eng.) Hamlyn, A. N.; James, O. (Royal Victoria Infirmary, Newcastle upon Tyne, England). *Gut* 18(11):A968-A969; 1977.

0651 SERUM IMMUNOGLOBULINS IN ALCOHOLIC LIVER DISEASE: CORRELATIONS WITH PORTAL HYPERTENSION, HEPATIC HISTOLOGY AND THERAPY [Abstract]. (Eng.) Maddrey, W. C.; Boitnott, J. K. (Johns Hopkins Univ. Sch. Medicine, Baltimore, MD). *Gastroenterology* 73(5):1232; 1977.

0652 LIVER REGENERATION AND HEPATROPHIC HORMONES IN MALNOURISHED ALCOHOLICS [Abstract]. (Eng.) Mahal, A.; Bakth, S.; Brody, D.; Leevy, C. M. (New Jersey Medical Sch., Newark, NJ). *Gastroenterology* 73(5):1233; 1977.

0653 EFFECT OF LOW SODIUM DIET (LSD) ON URINARY KALLIKREIN (UK) AND SODIUM (Na) EXCRETION IN PATIENTS WITH ASCITES FROM ALCOHOLIC LIVER DISEASE [Abstract]. (Eng.) Wong, P. Y.; Lipinski, T. L.; Capone, R. R. (Albany Medical Coll., Albany, NY 12208). *Gastroenterology* 73(5):1256; 1977.

0654 EFFECT OF ALBUMIN (A), SALINE INFUSION (SI) AND PERITONEOJUGULAR SHUNT (PJS) ON THE RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM IN PATIENTS WITH REFRACTORY ASCITES (RA) AND ALCOHOLIC LIVER DISEASE (ALD) [Abstract]. (Eng.) Wong, P. Y.; Sarfeh, I. J.; Lipinski, T. L.; Capone, R. R. (Albany Medical Coll., Union Univ., Albany, NY 12208). *Gastroenterology* 73(5):1256; 1977.

0655 EVIDENCE FOR THE PRESENCE OF PROLACTIN SECRETING ADENOMA IN SOME MEN WITH CIRRHOSIS AND GYNECOMASTIA [Abstract]. (Eng.) Van Thiel, D. H.; McClain, C. J.; Elson, M. K.; MacMillan, M. J.; Lester, R. (Dept. Medicine, Univ. Pittsburgh, Pittsburgh, PA). *Gastroenterology* 73(5):1251; 1977.

0656 ABNORMAL GROWTH HORMONE SECRETION IN MEN WITH LAENNEC'S CIRRHOSIS [Abstract]. (Eng.) Van Thiel, D. H.; Smith, W. I., Jr.; Wight, C. J.; Abuid, C. J. (Dept. Medicine, Univ. Pittsburgh, Pittsburgh, PA). *Gastroenterology* 73(5):1251; 1977.

0657 HEPATIC PROLYL HYDROXYLASE ACTIVITY AS A PARAMETER OF COLLAGEN SYNTHESIS AT INITIAL AND SUBSEQUENT BIOPSY OF PATIENTS WITH ALCOHOLIC LIVER DISEASE [Abstract]. (Eng.) Vidins, E. I.; Fox, S.; Fuller, G. C.; Kuttan, R.; Mann, S. W.; Rodil, J. V. (Dept. Pharmacology, Toxicology, Univ. Rhode Island, Kingston, RI). *Gastroenterology* 73(5):1252; 1977.

0658 HISTOLOGICAL AND ENDOSCOPIC ASPECTS OF ACUTE ALCOHOLIC HEPATITIS. (Spa.) Olivan, J.; Herrerias, J. M.; Perez-Cano, R.; Matilla, A.; Llamas, R.; Garrido, M. (Hospital Universitario de Sevilla, Seville, Spain). *Rev. Esp. Enferm. Apar. Dig.* 50(5):527-540; 1977.

See also, 0133, 0134, 0135, 0526, 0535, 0538, 0574, 0598, 0624, 0638, 0668, 0669, 0685.

Cirrhosis

0659 "TO SELECT BETTER--TO SHUNT BETTER." PREREQUISITES FOR A BETTER PORTACAVAL SHUNTING IN LIVER CIRRHOSIS. (Ger.) Thiel, H.; Wolter, J. (Med. Univ.-Klinik, Josef-Schneider-Strasse 2, 8700 Wurzburg, W. Germany). *Fortschr. Med.* 95(17):1159-1166; 1977.

Criteria that may improve the results of portacaval shunting in liver cirrhosis are discussed. Postoperative liver failure and portasystemic encephalopathy have frequently been reported after portacaval shunting; these may be due to a further reduction in hepatic blood flow, deprivation of the liver of portal venous blood supply with essential substances, and insufficient criteria for the selection of patients for this procedure. Better selection of patients is recommended, involving a complete evaluation of functional liver volume, the reaction of the hepatic artery to a portavenous increased or decreased blood flow, and new techniques to measure preoperative hemodynamics in liver cirrhosis (such as the transumbilical portal vein catheterization by means of a balloon catheter and transluminal portal vein injection with labeled microspheres for the determination of intrahepatic shunt volume). Differentiated shunt techniques (according to individual hemodynamic and functional data) may improve treatment results, especially for selective decompression and liver arterialization.

0660 LEFT SIDED SEGMENTAL PORTAL HYPERTENSION FOLLOWING MESOCAVAL INTERPOSITION SHUNT. (Eng.) Witte, C. L.; Ovitt, T. W.; Witte, M. H.; Clark, D. S. (Univ. Arizona Coll. Medicine, Tucson, AZ). *Surg. Gynecol. Obstet.* 145(2):169-174; 1977.

Experience with three cirrhotic patients in whom a mesocaval interposition shunt was carried out is presented. Following the procedure, bleeding esophageal varices recurred in two patients, and left sided portal hypertension partially persisted in one patient. Angiographic and pressure studies of the portal system demonstrated effective decompression of the greater splanchnic venous system but continued lesser splanchnic venous hypertension. Recurrent variceal hemorrhage ceased following emergency splenectomy. In contrast to what occurs following a standard portacaval shunt, after an interposition mesocaval shunt, altered jet streaming of mesenteric blood flow may divert gastrosplenic venous drainage away from the interposition shunt with persistence of lesser splanchnic venous hypertension. Recognition of this entity and of the need for splenectomy is advocated.

0661 CHRONIC HEPATIC ENCEPHALOPATHY. A PSYCHOMETRICAL STUDY. (Eng.) Rehnstrom, S.; Simert, G.; Hansson, J. A.; Johnson, G.; Vang, J. (Dept. Surgery, Univ. Lund, Lund, Sweden). *Scand. J. Gastroenterol.* 12(3):305-311; 1977.

The type and degree of intellectual impairment in hepatic encephalopathy was studied in 41 patients, and the influence of the liver disease etiology and of portasystemic shunts on this impairment was investigated. Psychometric tests were carried out in 41 patients with liver cirrhosis and suspected hepatic encephalopathy and compared with EEG examinations and clinical investigations. Marked intellectual impairment was noted frequently even when the clinical investigation was normal. This difference was mainly due to the preserved verbal ability of the patient. The etiology of the cirrhosis did not influence the test results. Male cirrhotic patients, with and without alcoholism, showed significantly more intellectual impairment than alcoholics without cirrhosis. Patients with portasystemic shunts showed only slightly reduced intellectual ability compared with those without shunts.

0662 RECTAL ^{13}N-AMMONIA TEST (^{13}N-LIVER/HEART RATIO), HEPATIC SINUSOIDAL PRESSURE AND PREVAILING PORTAL FLOW DIRECTION IN CIRRHOSIS OF THE LIVER. (Eng.) Hazenberg, H. J. A.; Gips, C. H.; Beekhuis, H.; Kruizinga, K. (Dept. Medicine, St. Jozefziekenhuis, Deventer 6600, Netherlands). *Acta Hepatogastroenterol. (Stuttg.)* 23(5):319-327; 1976.

Twelve of 26 patients with liver cirrhosis who had abnormally low 20-min liver/heart ^{13}N activity ratios (<2.25) were compared with cirrhotics who had normal ^{13}N liver/heart activity ratios after rectal administration of ^{13}N-ammonia in terms of several parameters relevant to liver cirrhosis. An abnormal conventional rectal arterial ammonia test (a rise of 17 μM ammonium ion, portasystemic shunts), an abnormally low urea index (1.2, prevailing hepatofugal portal venous flow direction), marked portal hypertension (hepatic sinusoidal pressure of >8 mm mercury), ascites, and extreme enlargement of the spleen occurred significantly ($p=0.04$) more often in patients with abnormally low ^{13}N liver/heart ratios than in those with ratios of 2.25 or greater. There was no correlation between the ^{13}N liver/heart ratio and the absence or presence of esophageal varices. The noninvasive rectal ^{13}N-ammonia test appears to be a convenient and informative test for the diagnosis of liver cirrhosis.

0663 RECTAL ADMINISTRATION OF ^{13}N-AMMONIA IN CIRRHOSIS OF THE LIVER. (Eng.) Hazenberg, H. J. A.; Gips, C. H.; Beekhuis, H.; Vaalburg, W. (Dept. Medicine, St. Jozefziekenhuis, Deventer 6600, Netherlands). *Acta Hepatogastroenterol. (Stuttg.)* 23(5):308-318; 1976.

^{13}N-activity was measured during 20-min periods in the liver, spleen, heart, lungs, and forearm of 16 controls and 26 cirrhotic patients after the

rectal administration of ^{13}N-labeled ammonium acetate solution. In all subjects, ^{13}N activity appeared in the liver 1 min after rectal administration, although, in some patients with cirrhosis, visualization of the liver was faint. At all times during the 20-min measurement period, liver activity was lower in cirrhotics than in controls ($p<0.01$). Liver/heart activity ratios were more discriminating between controls and cirrhotics ($p<0.05$ at all measurement times) than liver/lung and liver/spleen ratios. In controls, the 20-min liver/heart ratio was the most suitable ratio for determining the normal range (2.25-4.15). An abnormally low (<2.25) liver/heart ratio of ^{13}N activity at 20 min after rectal administration was observed in 12 of the 26 patients with cirrhosis. The advantages of the rectal ^{13}N-ammonia test are its noninvasiveness, giving no discomfort to the patient, the rapid disappearance of radioactivity, and the rapidity with which results are obtained.

0664 HEPATIC SUBCELLULAR DISTRIBUTION OF COPPER IN PRIMARY BILIARY CIRRHOSIS: COMPARISON WITH OTHER HYPERHEPATOCUPRIC STATES AND REVIEW OF THE LITERATURE. (Eng.) Owne, C. A., Jr.; Dickson, E. R.; Goldstein, N. P.; Baggenstoss, A. H.; McCall, J. T. (Mayo Clinic, Rochester, MN). *Mayo Clin. Proc.* 52(2):73-80; 1977.

The subcellular distribution of copper was studied in the liver of 17 normal adults, 1 newborn infant, and 35 patients with liver disease, including 4 with primary biliary cirrhosis. The findings were compared with those for the distribution of copper in the liver cell in normal, copper-laden, and copper-deficient adult rats, in newborn rats and pigs, and in adult pigs. A number of these livers were homogenized and divided into five particulate fractions, as well as the supernatant fraction, for copper analyses. Except in one category, it was found that the greater the total liver copper concentration, the greater the proportion in the larger subcellular particles and nuclei. The exception was patients with primary biliary cirrhosis and with very high total hepatic copper, with 45-75% of that copper residing in the supernatant fraction. In the seriously copper-deficient rat, the total hepatic copper was about half the normal amount. In this case, the shortage was reflected almost entirely in the supernatant fraction. By contrast, five patients with liver disease, who had hepatic copper concentrations below the normal range, had most of the copper in the supernatant fraction. It was also found that the copper content of blood exuding from the cut surfaces of the liver was about the same as the copper content of the liver itself.

0665 ESTRONE AND ESTRADIOL IN PATIENTS WITH CIRRHOSIS OF THE LIVER: EFFECTS OF ACTH AND DEXAMETHASONE. (Eng.) Kley, H. K.; Keck, E.; Kruskemper, H. L. (Medizinische Universitatsklinik, Moorenstrasse 5, 4000 Dusseldorf, W. Germany). *J. Clin. Endocrinol. Metab.* 43(3):557-560; 1976.

The influence of the adrenal gland on plasma estrogen levels was investigated in 12 men with hepatic cirrhosis and in 12 healthy male controls. Cirrhotic patients showed normal base cortisol levels (122 ± 41 ng/ml) compared with controls (122 ± 41 ng/ml). Under basal conditions, cirrhotic patients showed moderately higher ($p<0.05$) plasma estradiol levels (36.0 ± 4.7 pg/ml) and considerably higher ($p<0.01$) plasma estrone levels (67.8 ± 20.8 pg/ml) compared with controls (28.5 ± 5.4 and 32.5 ± 5.6 pg/ml for estradiol and estrone, resp.). The administration of ACTH (1 mg, i.v.) significantly ($p<0.01$) increased cortisol and estrone levels to values of 434 ± 68 ng/ml and 139.6 ± 27.5 pg/ml, resp., in cirrhotics and values of 412 ± 63 ng/ml and 59.8 ± 8.5 pg/ml, resp., in controls. Estradiol levels did not significantly change during ACTH stimulation in either group. Adrenal gland suppression with dexamethasone (1.5 mg, p.o., 3 times/day) for 5 days significantly reduced estrone and estradiol levels from basal values of 72.6 ± 26.8 pg/ml and 35.5 ± 2.1 pg/ml, resp., to values of 37.1 ± 17.6 ($p<0.05$) and 23.9 ± 3.6 ($p<0.001$), resp., in cirrhotics and from basal values of 33.3 ± 6.5 pg/ml and 25.5 ± 4.8 pg/ml, resp., to values of 12.0 ± 4.0 ($p<0.001$) and 15.9 ± 4.7 ($p<0.01$), resp., in controls. The absolute decrease of estrone was significantly greater ($p<0.005$) in cirrhotics than in controls, although suppressed plasma levels of estrone, as well as of estradiol, were still higher ($p<0.05$) than those of controls. These findings indicate that the elevated plasma estrone levels in cirrhotic patients are predominantly due to adrenal production and that peripheral conversion of androgens to estrone rather than to estradiol is more effective in sustaining plasma estrogen levels in this disorder.

0666 ENDOTOXAEMIA AND RENAL FAILURE IN CIRRHOSIS AND OBSTRUCTIVE JAUNDICE. (Eng.) Wilkinson, S. P.; Moodie, H.; Stamatakis, J. D.; Kakkar, V. V.; Williams, R. (King's Coll. Hosp. and Medical Sch., London SE5 8RX, England). *Br. Med. J.* 2(6049):1415-1418; 1976.

The possible role of endotoxins in the pathogenesis of renal failure was investigated in 45 patients with cirrhosis and 12 with obstructive jaundice. Twenty-five of the 45 cirrhotic patients had renal failure, and 21/25 had endotoxemia. None of these 21 patients had other evidence of gram-negative infection at the time of the study, and the endotoxemia was therefore probably due to impaired hepatic clearance of toxins normally absorbed from the gastrointestinal tract. In contrast, bacteriological evidence of gram-negative infection was found in four of six patients with obstructive jaundice and endotoxemia. Only 2/20 cirrhotics without renal failure had endotoxemia, and there was a highly significant difference ($p<0.001$) between this group and the renal failure patients. None of the six patients with obstructive jaundice and normal renal function had endotoxemia. The results indicate that there is

a close relation between endotoxemia and renal failure in liver diseases.

0667 IMMUNE RESPONSE TO ϕX 174 IN MAN. 5. PRIMARY AND SECONDARY ANTIBODY PRODUCTION IN PRIMARY BILIARY CIRRHOSIS. (Eng.) Thomas, H. C.; Holden, R.; Jones, J. V.; Peacock, D. B. (Royal Free Hosp., Hampstead, London N.W. 3, England). *Gut* 17(11):844-848; 1976.

The primary and secondary immune response to an i.v. antigenic challenge with the bacteriophage ϕX 174 was studied in 12 patients with primary biliary cirrhosis. Patients were injected with 2.0 x 10^9 plaque-forming units (PFU) of ϕX 174, and blood samples were collected for 28 days. A second injection was given between the 28th and 42nd days, and blood samples were collected for one month. All patients had a normal immunological response to the first injection. After the second injection, four patients had a normal response, and eight demonstrated a marked reduction in IgG and in the concentration of peripheral T cells. The ratio of serum IgM to IgG was significantly ($p<0.25$) increased in the eight patients with impaired immunological response after the second injection. There was no correlation between the degree of alteration of the secondary immune response to ϕX 174 and the severity of the liver cirrhosis, as shown by degree of cholestasis or hepatocellular necrosis.

0668 HEPATIC AND EXTRAHEPATIC COPPER CONCENTRATIONS IN CIRRHOSIS OF THE LIVER [Abstract]. (Eng.) Ritland, S.; Johansen, O.; Steinnes, E. (Natl. Hosp. Norway, Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):77; 1977.

0669 SYSTEMIC AVAILABILITY OF LIDOCAINE IN PATIENTS WITH LIVER DISEASE [Abstract]. (Eng.) Tschanz, C.; Steiner, J. A.; Hignite, C. E.; Huffman, D. H.; Azarnoff, D. L. (Univ. Kansas Medical Center, Kansas City, MO). *Clin. Res.* 25(4):609A; 1977.

0670 CERVICAL LYMPHATO VENOUS SHUNT IN TREATMENT OF ASCITES IN CAVAL CONSTRICTED DOGS AND IN PATIENTS WITH HEPATIC CIRRHOSIS. EXPERIMENTAL OBSERVATIONS AND 7 YEARS CLINICAL EXPERIENCE. (Eng.) Serenyi, P.; Magyar, Z.; Szabo, G. (Natl. Inst. Traumatology, Budapest, Hungary). *Lymphology* 9(2):53-61; 1976.

0671 A DOUBLE BLIND CONTROLLED STUDY OF CHOLANGIOGRAPHIC CONTRAST AGENTS IN CIRRHOTIC PATIENTS [Abstract]. (Eng.) Bar-Meir, S.; Ramsby, G. R.; Atterbury, C. E.; Conn, H. O. (Veterans Admin. Hosp., West Haven, CT). *Gastroenterology* 73(5):1212; 1977.

0672 PORTAL HYPERTENSION IN CHILDREN [Abstract]. (Eng.) da Silveira Montenegro, T.; Licht da Silva, G.; Zaslavsky, C.; Pereira Lima, J.; Antunes, C. R.; Almeida, H. (No affiliation given). *Arq. Gastroenterol.* 14(3):161; 1977.

0673 PRESENTING CLINICAL FEATURES OF HBsAg-POSITIVE LIVER CIRRHOSIS IN CHINESE [Abstract]. (Eng.) Lam, K. C.; Lai, C. L.; Lee, A. (Queen Mary Hosp., Hong Kong). *Gastroenterology* 73(5):1230; 1977.

0674 THE ROLE OF ULTRASONOGRAPHY IN THE DIAGNOSIS OF CIRRHOSIS [Abstract]. (Eng.) Lemon, S. K.; Gosink, B. B.; Scheible, F. W. (Veterans Admin. Hosp., San Diego, CA). *Gastroenterology* 73(5):1231; 1977.

0675 ABSORPTION AND HYDROXYLATION OF VITAMIN D IN PRIMARY BILIARY CIRRHOSIS (PBC) [Abstract]. (Ger.) Krawitt, E. L.; Grundman, M. J.; Mawer, E. B. (Dept. Medicine, Univ. Manchester, Manchester, England). *Gastroenterology* 73(5): 1229; 1977.

0676 TRANSITION TO HIGH PROTEIN CIRRHOTIC ASCITES DURING DIURESIS [Abstract]. (Eng.) Hoefs, J. (Univ. Southern California Sch. Medicine, Los Angeles, CA). *Gastroenterology* 73(5):1225; 1977.

0677 EXTRACTS OF ^{125}I ALBUMIN MICROAGGREGATES FROM PORTAL VEIN (PV) AND HEPATIC ARTERY (HA) BLOOD: AN INDEX OF FUNCTIONAL HEPATIC BLOOD SUPPLY IN CIRRHOTICS [Abstract]. (Eng.) Huet, P. M.; DuReau, A.; Marleau, D. (Hopital Saint-Luc, Montreal, Canada). *Gastroenterology* 73(5):1226; 1977.

0678 FUNCTIONAL PORTAL BLOOD SUPPLY: ITS RELATIONSHIP TO 99mTc PHYTATE EXTRAHEPATIC UPTAKE [Abstract]. (Eng.) Huet, P. M.; Marleau, D.; Chartrand, R. (Hopital Saint-Luc, Montreal, Canada). *Gastroenterology* 73(5):1226; 1977.

0679 THE HAEMODYNAMIC AND RENAL EFFECTS OF THE PERITONEOJUGULAR (LEVEEN) SHUNT [Abstract]. (Eng.) Greig, P. D.; Langer, B.; Ruse, J.; Wong, P. Y.; Blendis, L. M. (Dept. Medicine, Univ. Toronto, Toronto, Canada). *Gastroenterology* 73(5):1222; 1977.

0680 CIRCULATING IMMUNE COMPLEXES AND AUTOIMMUNE FEATURES IN PRIMARY BILIARY CIRRHOSIS: IMPLICATIONS FOR PATHOGENESIS [Abstract]. (Eng.) Gupta, R. C.; Dickson, E. R.; McDuffie, F. C. (Mayo Clinic, Rochester, MN). *Gastroenterology* 73(5):1223; 1977.

0681 LIVER SURFACE CHARACTERISTICS AS OBSERVED DURING LAPAROSCOPY CORRELATED TO THE

HISTOLOGICAL FINDINGS OF THE BIOPSY [Abstract]. (Eng.) Heit, H. A.; Johnson, L. F.; Rabin, L. (Walter Reed Army Medical Center, Washington, DC). *Gastroenterology* 73(5):1224; 1977.

0682 THE SERIOUS COMPLICATIONS OF DIURESIS IN THE ASCITIC PATIENT: DOES THE DIURETIC REGIMEN MATTER [Abstract]? (Eng.) Fogel, M. R.; Knauer, C. M.; Sawhney, V.; Miller, R.; Gregory, P. B. (Dept. Medicine, Stanford Univ., Stanford, CA 94305). *Gastroenterology* 73(5): 1220; 1977.

0683 AFFLUENCE, ALCOHOL, AND CIRRHOSIS [Abstract]. (Eng.) Galambos, J. T. (Emory Univ., Atlanta, GA). *Gastroenterology* 73(5):1221; 1977.

0684 PARENTERAL 1,25-DIHYDROXYCHOLECALCIFEROL IN HEPATIC OSTEOMALACIA [Abstract]. (Eng.) Long, R. G.; Varghese, Z.; Meinhard, E.; Skinner, R. K.; Wills, M. R.; Sherlock, S. (Royal Free Hosp., Pond St., Hampstead, London, England). *Gut* 18(11): A954; 1977.

0685 BILIARY AND FAECAL BILE ACID COMPOSITION IN LIVER DISEASE [Abstract]. (Eng.) Krasner, N.; Atherton, S. T.; Nelson, L. M.; Fleming, H.; Russell, R. I. (Royal Infirmary, Glasgow, Scotland). *Gut* 18(11):A988; 1977.

0686 C1q BINDING ACTIVITY AND COMPLEMENT (C1q, C3) CATABOLISM IN PRIMARY BILIARY CIRRHOSIS AND CHRONIC ACTIVE LIVER DISEASE [Abstract]. (Eng.) Thomas, H. C.; Potter, B. J.; Elias, E.; De Villiers, D.; Sherlock, S. (Royal Free Hosp., Hampstead, London, England). *Gut* 18(11):A979; 1977.

0687 NAFCILLIN DISPOSITION IN HEPATOBILIARY DISEASE [Abstract]. (Eng.) Marshall, J. P.; Salt, W. B.; Elam, R. O.; Wilkinson, G. R.; Schenker, S. (Vanderbilt Univ. Sch. Medicine, Nashville, TN). *Clin. Res.* 25(4):608A; 1977.

See also, 0174, 0288, 0535, 0538, 0552, 0553, 0563, 0566, 0574, 0575, 0577, 0580, 0629, 0636, 0638.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

0688 CRITICAL EVALUATION OF PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY. (Ger.) Gattinger, A.; Schmoller, H.; Suwandschieff, N. (Rontgendiagnostisches Zentralinstitut des Landeskrankenanstalten, Mullner Hauptstrasse 48, A-5020 Salzburg, Austria). *Wien. Med. Wochenschr.* 127(7): 218-221; 1977.

The results of percutaneous transhepatic cholangiography (PTC) in 33 patients with suspected bile duct obstruction are presented. Puncture of the ducts was impossible in five cases. The cholangiogram was unremarkable in three cases. Bile duct obstruction due to malignant tumor was diagnosed in 15 cases; all these cases were verified at surgery. The tumors included carcinomas of the gallbladder, head of the pancreas, and of the ampulla of Vater. The cholangiogram showed obstruction due to stones in six cases; the diagnosis was correct in five of six cases, while carcinoma of the head of the pancreas was found in the sixth. Cicatricial stenosis was diagnosed in four cases; this diagnosis was correct in two cases, while recurrent tumor and sandpaper gallbladder were found at surgery in the other two cases. The rate of correct cholangiographic diagnosis was 85.7%. The findings indicate the value of percutaneous transhepatic cholangiography in the diagnosis of obstructive jaundice. This test should not be performed on inoperable patients, nor in those with severe disturbance of blood clotting.

0689 ENDOSCOPIC PAPILLOTOMY (EPT). (Eng.) Manegold, B. C. (Klinikum Mannheim der Universitat Heidelberg, Mannheim, W. Germany). *Chir. Gastroenterol.* 10(4):433-435; 1976.

Experience with endoscopic papillotomy for the treatment of papillary stenosis and small common bile duct stones is presented. This procedure was indicated for common bile duct stones in 58 patients, for papillary stenosis in 3, and for a rare case of obstructive jaundice caused by a foreign body having moved from its bed in the liver into the main bile duct in 1. Endoscopic papillotomy was successful in 55 cases, and complete discharge of concrements was proved in 48. In four cases, papillotomy was successful, but concrements were left behind, making choledochotomy necessary. The procedure was

complicated by perforation in two cases, by pancreatitis in three, and by cholangitis in one; these patients were treated conservatively, and all survived. Papillotomy was unsuccessful in seven cases. The papilla was not cannulated in four cases; a juxtapapillary diverticulum was present in one case; and technical difficulties occurred in two cases. Endoscopic papillotomy is an effective, low-risk method for the treatment of papillary stenosis and small common bile duct stones in aged patients. Its success depends upon the length of the incision, the diameter of the stone, and the anatomical form of the prepapillary duct.

0690 ANAEROBIC BACTERIA IN BILIARY DISEASE IN ELDERLY PATIENTS. (Eng.) Shimada, K.; Inamatsu, T.; Yamashiro, M. (Tokyo Metropolitan Geriatric Hosp., 35-2, Sakae-cho, Itabashi-ku, Tokyo 173, Japan). *J. Infect. Dis.* 135(5):850-854; 1977.

Gallbladder bile from 52 elderly subjects (mean age 73.4 yr) who had undergone biliary tract surgery was examined for the presence of bacteria. Thirty-three patients had not been given antibiotics before their operations. Twelve patients had sterile bile, 18 specimens of bile yielded anaerobes as well as aerobes, and 22 yielded aerobic bacteria only. *Escherichia coli* was the most commonly isolated organism (30 strains). *Bacteroides fragilis* was the most frequently encountered anaerobic bacterium and was found in 15 patients. The *Klebsiella-Enterobacter* group was the second most commonly isolated group and *B. fragilis* was third. *Clostridium perfringens* was recovered in 10 bile specimens. Anaerobic bacteria were recovered more frequently in patients with ductal obstruction. The relatively frequent isolation of anaerobes, especially of *B. fragilis*, may be related to the anaerobic techniques used, to the age of the patients, and to the high incidence of pigment stones among the subjects (36/39 patients with cholelithiasis).

0691 BILIARY LIPID SECRETION IN CHOLESTEROL GALLSTONE DISEASE: THE EFFECT OF CHOLECYSTECTOMY AND OBESITY. (Eng.) Shaffer, E. A.; Small, D. M. (Montreal General Hosp., 1650 Cedar Ave., Montreal, Quebec, H3G 1A4, Canada). *J. Clin. Invest.* 59(5):828-840; 1977.

To define the biliary secretory mechanism(s) responsible for the production of abnormal bile leading to cholesterol gallstone disease, the rate of biliary lipid secretion was measured by a duodenal marker perfusion technique, and the bile salt pool was estimated simultaneously by isotope dilution. Two groups of control patients expected to have a normal biliary lipid composition (14 without hepatobiliary disease and 6 with pigment gallstones) were compared with two groups expected to have abnormal bile (10 nonobese patients with cholesterol gallstones and 7 obese subjects without gallstones). Both control groups had nearly identical biliary lipid secretion rates and a corresponding low relative molar concentration of cholesterol. Two different secretory mechanisms were responsible for the abnormal bile in the experimental groups. In the nonobese patients with cholesterol gallstones, bile salt and phospholipid secretion rates were reduced significantly. Conversely, the grossly obese subjects had an increased cholesterol secretion. To determine how cholecystectomy improves biliary lipid composition, three groups of gallstone patients (6 with pigment stones, 4 grossly obese with cholesterol stones, and 13 nonobese with cholesterol stones) were examined after full recovery from surgery. In the nonobese patients with cholesterol gallstones, bile salt and phospholipid secretion increased significantly, causing a definite improvement in bile composition. Cholecystectomy produced a similar but less marked trend in the obese patients with cholesterol stones and in the patients with pigment stones. Cholesterol secretion, however, was unaffected by surgery. The bile salt pool was small in the nonobese patients with cholesterol gallstones, and it became slightly smaller after cholecystectomy. The pool was significantly reduced by cholecystectomy in the pigment stone patients and in the obese patients with cholesterol gallstones. Cholecystectomy in all three groups caused a greater fraction of the pool to cycle around the enterohepatic circulation each hour. This more rapid cycling increased bile salt and phospholipid secretion, and was responsible for the improved composition found after cholecystectomy.

0692 FROM MICROLITH TO GALLSTONE. (Eng.) Van de Wal, J. W. M. (St. Elisabeth Ziekenhuis, Amersfoort, Netherlands). *Tijdschr. Gastroenterol.* 20(2):101-112; 1977.

The relationship between gallstone type and gallbladder histopathology was examined in an unselected series of 120 cholecystectomized patients. Among 14 patients with normal gallbladder histology, cholesterol microliths were found in 8, cholesterol mulberry calculi in 3, and other types of calculi in 3. In 11 patients with hypertrophy and hyperplasia of the gallbladder musculature, cholesterol microliths were found in 2, cholesterol mulberry calculi in 2, and other types of calculi in 7. Cholesterol microliths were found in 1 patient, cholesterol mulberry calculi in 4, and other types of calculi in 6 among 11 patients with minimal signs of gallbladder inflammation. In this latter group, there was an increase in the faceting and deposits of gallstones. Among 84 patients with signs of acute-chronic gallbladder inflammation, cholesterol microliths were found in 5, cholesterol mulberry calculi in 4, and calculi of all sizes, shapes, colors, and compositions in 75. As the condition of the gallbladder progresses from normal to pathological, the proportion of cholesterol microliths decreases in relation to other types of calculi. Muscular hypertrophy and hyperplasia may constitute the first visible alteration of the gallbladder wall during gallstone formation.

0693 OCCURRENCE OF DISSEMINATED INTRAVASCULAR COAGULATION (DIC) IN OBSTRUCTIVE JAUNDICE AND ITS RELATION TO BILIARY TRACT INFECTION. (Eng.) Takeda, S.; Takaki, A.; Ohsato, K. (Kyushu Univ. Faculty Medicine, Fukuoka, Japan). *Jpn. J. Surg.* 7(2):82-89; 1977.

Coagulation studies were done on 78 consecutive cases of obstructive jaundice with or without biliary tract infection to emphasize the significant role of biliary tract infection in the development of disseminated intravascular coagulation (DIC). Among 26 patients with biliary tract infection, 6 patients developed diffuse bleeding tendency (ecchymoses, gastrointestinal bleeding, hematuria, and bleeding from operative wounds, choledochostomies, and venipuncture sites. Supprative cholangitis occurred in all cases. The signs of multiorgan dysfunction were seen in most patients, including oliguria or anuria (3 patients), cardiac symptoms (2), respiratory distress (5), and mental confusion (4). Hypotension was present in five patients. Most patients showed marked coagulation defects characterized by thrombocytopenia, decreased fibrinogen and antithrombin III levels ($p<0.05$), decreased plasminogen levels ($p<0.02$), narrowing of the maximal amplitude in thrombelastograms, increase of fibrin degradation products ($p<0.05$), and increase of positive soluble fibrin monomer complexes. Five patients died from 10 days to 3 months after the biliary tract decompression. In two cases, postmortem examination revealed multiple fibrin thrombi in lungs and other organs. The remaining 20 patients showed no bleeding tendency but remarkable hypercoagulability with decreased fibinolytic activity. The development of DIC was more frequent in cases in which biliary tract decompression failed. These clinical, hematological, and pathological findings strongly suggest that the development of bleeding tendency in obstructive jaundice may be due to the occurrence of DIC, i.e., that hypercoagulability caused by the biliary tract infection is responsible.

0694 FUNCTION CHANGES OF THE SPHINCTER OF ODDI AFTER DIFFERENT TYPES OF VAGOTOMY. (Eng.) Schumann, Z.; Darup, K.; Gentsch, H. (Univ. Erlangen, Dept. Surgery, Nurnberg, W. Germany). *Chir. Gastroenterol.* 10(4):437-439; 1976.

Myoelectric changes in the papilla of Vater were studied in 24 dogs after truncal, selective, or parietal cell vagotomy. In six dogs that underwent truncal vagotomy, there was an initial decrease in tone in the sphincter of Oddi; this tone later increased and approached the initial basal level. The amplitude of the action potential was increased by 150 ± 10 μV per unit, and frequency was decreased to 72 Hz. The extrahepatic bile ducts appeared atonic and dilated. After selective vagotomy, the amplitude was similar to that in nonoperated controls; the frequency was 72 Hz. There were no changes in the electromyographic activity of the papilla of Vater in the dogs that underwent parietal cell vagotomy; the amplitude and frequency were 22 ± 5 μV/U and 100 Hz, resp., values identical with those of the controls. These results indicate that parietal cell vagotomy is the procedure of choice in the treatment of duodenal ulceration.

0695 ENDOSCOPIC PAPILLOTOMY--COMPLICATIONS--RESULTS [Abstract]. (Eng.) Rosseland, A. R.; Valnes, K. (Central Hosp. of Akershus, N-1474 Nordbyhagen, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):85; 1977.

0696 ENDOSCOPIC PAPILLOTOMY IN PATIENTS WITH A BILLROTH-II PARTIAL GASTRECTOMY [Abstract]. (Eng.) Rosseland, A. R. (Centralhosp. of Akershus, N-1474 Nordbyhagen, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):84; 1977.

0697 TREATMENT OF CHOLEDOCHOLITHIASIS BY ENDOSCOPIC PAPILLOTOMY, RE-PAPILLOTOMY AND ENDOSCOPIC CHOLEDOCHODUODENOSTOMY THROUGH CHOLEDOCHODUODENAL FISTULA [Abstract]. (Eng.) Osnes, M. (Ulleval Hosp., Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):66; 1977.

0698 AN ASSESSMENT OF INTRAVENOUS CHOLANGIOGRAPHY (IVC) AND ENDOSCOPIC RETROGRADE CHOLANGIOGRAPHY (ERC) IN THE DIAGNOSIS OF CHOLELITHIASIS [Abstract]. (Eng.) Nordshus, T.; Lowe, P. P.; Osnes, M. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):64; 1977.

0699 ERCP AND/OR PTC IN SUSPECTED OBSTRUCTIVE JAUNDICE. REPORT FROM AN ONGOING PROSPECTIVE STUDY [Abstract]. (Eng.) Matzen, P.; Christensen, J. H.; Haubek, A. (Hvidovre Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12 (Suppl. 45):60; 1977.

0700 BACTERIOLOGICAL STUDIES OF CHOLEDOCHAL BILE IN PATIENTS WITH GALLSTONES AND JUXTA-PAPILLARY DUODENAL DIVERTICULA [Abstract]. (Eng.) Lotveit, T.; Osnes, M.; Aune, S. (Ulleval Hosp., Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):58; 1977.

0701 BENIGN POLYPOID TUMORS IN THE COMMON BILE DUCT DEMONSTRATED BY ERCP [Abstract]. (Eng.) Kruse, A.; Mikkelsen, K.; Kraglund, K. (Aarhus Kommunehosp., DK-8000 Aarhus C, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45): 54; 1977.

0702 STONE IN BILE DUCT WITH NEGATIVE CHOLANGIOPANCREATOGRAPHY [Letter to Editor]. (Eng.) Benson, E. A. (Leeds General Infirmary, Leeds LS1 3EX, England). *Lancet* 2(8051):1292; 1977.

0703 VALUE OF TRANSJUGULAR CHOLANGIOGRAPHY FOR THE OPERATIVE EVALUATION OF OBSTRUCTIVE

JAUNDICE [Abstract]. (Eng./Ger.) Thelen, M.; Janson, R.; Schulz, D.; Paquet, K. J. (Radiologische Klinik der Universitat, D-5300 Bonn-Venusberg, W. Germany). *Langenbecks Arch. Chir.* 345:582; 1977.

0704 LATE RESULTS OF MORE THAN 300 BILIODIGESTIVE ANASTOMOSES CAUSED BY TRAUMA OR STRICTURE OF THE BILE DUCT [Abstract]. (Eng./Ger.) Paquet, K. J.; Gutgemann, A. (Chirurgische Universitatsklinik, D-5300 Bonn-Venusberg, W. Germany). *Langenbecks Arch. Chir.* 345:581; 1977.

0705 PROBLEMS OF CONSERVATIVE DISSOLUTION OF GALLSTONES FROM A SURGICAL VIEWPOINT [Abstract]. (Eng./Ger.) Franke, S.; Winter, G.; Klee, W. E. (Chirurgische Universitatsklinik, Josef Schneider-Str. 2, D-8700 Wurzburg, W. Germany). *Langenbecks Arch. Chir.* 345:583; 1977.

0706 OPERATIVE BILIARY ENDOSCOPY [Abstract]. (Eng./Ger.) Lennert, K. A. (Evang. Krankenhaus, Virchowstr. 20, D-4200 Oberhausen/Rhld., W. Germany). *Langenbecks Arch. Chir.* 345:582; 1977.

0707 HIDA SCANNING AS A DIFFERENTIAL DIAGNOSTIC AID IN JAUNDICED PATIENTS [Abstract]. (Eng.) Schoubye, J.; Pedersen, S. A.; Jorgensen, E. O. (Odense Univ. Hosp., Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):90; 1977.

0708 VALUE OF ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY (ERCP) [Abstract]. (Eng.) Ruud, T. E. (Telemark Sentralsjukehus, Skien, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):87; 1977.

0709 GREY SCALE ULTRASONOGRAPHY (U.S.) AND ENDOSCOPIC RETROGRADE PANCREATOGRAPHY (ERCP) IN PATIENTS WITH OBSTRUCTIVE JAUNDICE [Abstract]. (Eng.) Hoiseth, A.; Osnes, M.; Swensen, T. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):38; 1977.

0710 THE SIGNIFICANCE OF ENDOSCOPIC RETROGRADE CHOLANGIOGRAPHY (ERC) IN PATIENTS ADMITTED FOR BILIARY TRACT SURGERY [Abstract]. (Eng.) Havig, O.; Osnes, M.; Aune, S. (Ulleval Hosp., Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):33; 1977.

0711 BILE DUCT CARCINOMA ASSOCIATED WITH ULCERATIVE COLITIS. (Eng.) Akwari, O. E.; Van Heerden, J. A.; Adson, M. A.; *et al.* (Mayo Clinic Foundation, Rochester, MN). *Rev. Surg.* 33(4):289-293; 1976.

0712 LEIOMYOMA OF THE COMMON BILE DUCT CAUSING OBSTRUCTIVE JAUNDICE. (Eng.) Kune, G. A.; Polgar, V. (Royal Melbourne Hosp., Melbourne, Australia). *Med. J. Aust.* 1(19):698-699; 1976.

0713 JAUNDICE IN SEVERE BACTERIAL INFECTION. (Eng.) Miller, D. J.; Keeton, G. R.; Webber, B. L.; Saunders, S. J. (Univ. Cape Town Liver Res. Group, Univ. Cape Town, Cape Town, South Africa). *Gastroenterology* 71(1):94-97; 1976.

0714 BENIGN BILIARY TRACT DISEASE IN THE PATIENT OVER 80. (Eng.) McKirdie, M. (Good Samaritan Hosp. Medical Center, Portland, OR). *Am. Surg.* 42(7):471-473; 1976.

0715 THE RESULTS OF COMMON DUCT SURGERY. (Fre.) Kayabali, I.; Ceylan, I.; Aras, N. (Faculty Medicine, Anakara, Turkey). *Lyon Chir.* 72(4):251-253; 1976.

0716 ENDOSCOPIC PAPILLOTOMY AS TREATMENT OF CHOLEDOCHOLITHIASIS [Abstract]. (Eng.) Kruse, A. (Aarhus Kommunehospital, DK-8000 Arhus C, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):51; 1977.

See also, 0150, 0256, 0379, 0389, 0438, 0513, 0535, 0575, 0576, 0624, 0671, 0687, 0754.

GENERAL

0717 ALIMENTARY TRACT MANIFESTATIONS OF MULTIPLE ENDOCRINE NEOPLASIA, TYPE 2b. (Eng.) Carney, J. A.; Hayles, A. B. (Mayo Clinic, Rochester, MN). *Mayo Clin. Proc.* 52(9):543-548; 1977.

Alimentary tract manifestations were found in all of 17 patients with multiple endocrine neoplasia, type 2b, and the cases of 7 patients are presented. The alimentary tract manifestations were important because (a) they were chronic; (b) they were severe and led to abdominal operation in 5 patients; (c) they antedated detection of the endocrine neoplasms in the syndrome in 16 patients (94%); and (d) they provided clinical clues that stimulated

a search for thyroidal C-cell and adrenal medullary disease in 6 patients. The alimentary tract manifestations were diverse; the symptoms included constipation, diarrhea, difficulty with feeding, projectile vomiting, crampy abdominal pain, and loud borborygmi. Other findings included thickened lips, nodules on the anterior third of the tongue, abdominal distention, visible peristaltic waves, and roentgenographic evidence of megacolon or diverticulosis of the colon or of dilatation of the small intestine and stomach. Initial misinterpretation or failure to realize the significance of one or more of these alimentary tract manifestations led to suspicion of aganglionic megacolon (3 patients), malabsorption syndrome (2 patients), and tracheal ring (1 patient).

0718 IMMUNOGLOBULINS IN JEJUNAL MUCOSA AND SERUM FROM PATIENTS WITH DERMATITIS HERPETIFORMIS. (Eng.) Baklien, K.; Fausa, O.; Thune, P. O.; Gjone, E. (Rikshospitalet, Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(2):161-168; 1977.

The quantitative distribution of immunoglobulin (Ig)-producing cells was determined by paired immunohistochemistry in the proximal jejunal mucosa of 29 patients with dermatitis herpetiformis (DH). The specimens were categorized stereomicroscopically into three groups on the basis of mucosal abnormality. Compared with control subjects, the total number of Ig-producing cells in a defined "mucosal tissue unit" was raised by a factor of 1.4 in group I (normal or minor abnormality), but the class ratios remained normal. In group III (major abnormality), the total immunocyte number was raised 2.6 times; IgA, IgM, and IgG cells showed a 2.4-, 3.5-, and 5.1-fold increase, resp. Group II (intermediate abnormality) fell between the two other groups with regard to changes in immunocyte population. The local immunocyte pattern was very similar to that previously reported for celiac disease (CD) specimens with comparable mucosal abnormalities. There were few IgD cells, and there was no increase in these cells, compared with control levels. IgE cells were also infrequent, but they were more numerous than in control tissue. As in CD, staining for extracellular Ig in the lamina propria tended to be more intense than that in controls. Normal or increased staining for SC, IgA, and IgM in the crypt epithelium indicated a normal external transfer of these components. DH differed from CD in showing no significant increase in serum IgA level. Serum IgM was reduced in group III, and IgG was increased in group I.

0719 CONGENITAL CHLORIDE DIARRHOEA. (Eng.) Holmberg, C.; Perheentupa, J.; Launiala, K.; Hallman, N. (Children's Hosp., Univ. Helsinki, Helsinki, Finland). *Arch. Dis. Child.* 52(4):255-267; 1977.

Clinical findings in 21 Finnish children with congenital chloride diarrhea are reported. All children were born 1-8 weeks prematurely. Hydramnios was present in all cases, and no meconium was seen; thus, the onset of diarrhea occurred *in utero*. Diarrhea or passing of large volumes of urine was noted on the first day of life, and the abdomen was usually large and distended. The abnormally large neonatal weight was associated with hypochloremia and hyponatremia. A few infants survived the neonatal period without adequate therapy, but later they developed hypochloremia, hypokalemia, and metabolic alkalosis associated with hyperaldosteronism. However, these conditions may be absent; therefore, diagnosis is based on a history of hydramnios and diarrhea and a fecal Cl^- concentration >90 mM. Lower fecal Cl^- concentrations were seen only in chronic hypochloremia, which is also associated with achloriduria. Adequate treatment consists of full continuous replacement of the fecal losses of water, NaCl, and KCl. These substances should be given i.v. in the early neonatal period; later, they can be given p.o., with meals. The electrolyte dose has to be adjusted to maintain normal serum concentrations, normal blood pH, and some chloriduria. This therapy prevents the renal lesions and the retarded growth and psychomotor development that were seen in children who were diagnosed late and in those who received inadequate replacement therapy. The watery diarrhea persists and increases slightly with age, although patients learn to live with their disease and to make an adequate social adjustment.

0720 SPECIAL O:K:H SEROTYPES AMONG ENTEROTOXIGENIC E. coli STRAINS FROM DIARRHEA IN ADULTS AND CHILDREN: OCCURRENCE OF THE CF (COLONIZATION FACTOR) ANTIGEN AND OF HEMAGGLUTINATING ABILITIES. (Eng.) Orskov, I.; Orskov, F. (Collaborative Center for Reference and Res. on *Escherichia* [WHO], Statens Seruminstitut, Amager Blvd. 80, DK-2300 Copenhagen S, Denmark). *Medical Microbiol. Immunol. (Berl).* 163(2):99-110; 1977.

Countercurrent immunoelectrophoresis was used to determine polysaccharide K antigens in O:H serotypes among 77 enterotoxigenic *Escherichia coli* strains, including strains from human diarrhea. Members of each O:H serotype had the same type of K antigen and were characterized by a certain fermentation pattern. Some O:H serotypes had no K antigen. The serofermentative types defined were: O6:K15:H16, O8:K40:H9, O15:H11, O25:K7:H42, O25:K98:H^-, O78:H11, O78:H12, and O149:H10. Some strains of serotype O149:H10, which are suspected of being associated with foodborne infections, had K88 antigen. This serotype belongs to the group of strains causing diarrhea in swine. Colonization factor (CF), a pilus-like antigen that is plasmid-controlled and that endows the bacterial cells with an ability to colonize the intestine, was found only in human enterotoxigenic strains belonging to O group 78. In particular, CF was found in 17 of 77 enterotoxigenic strains, and all 17 belonged to O78:H11, O78:H12, or O78:H^-; CF was not found among 248 type O78 strains from sources other than human diarrhea. The presence of CF in type O78 strains was correlated with the presence of a mannose-resistant ability to cause agglutination of human RBC. An examination of the hemagglutinating ability of the other serotypes revealed that O25:K7:H42 strains were similar to the O78 strains in this respect.

0721 EPITHELIOID GERMINAL CENTERS: AN ACQUIRED IMMUNOLOGIC DEFICIT? (Eng.) Millikin, P. D. (Peoria-Tazewell Pathology Group, S.C., 5409 North Knoxville, Peoria, IL 61614). *Am. J. Clin. Pathol.* 67(6):545-549; 1977.

In an attempt to determine whether the fatal outcome of childhood infections associated with epithelioid germinal center abnormalities is related to an acquired immunologic deficit, the histologic structure of lymphoid tissue in cases of fatal infection is reviewed. Epithelioid germinal centers, completely devoid of the usual dark zone of rapidly dividing blast cells, were found in 16 infants and children, one of whom also had Reye's syndrome. In four additional cases, there was toxic follicle alteration, or massive necrosis of germinal center cells. Toxic follicle alteration and epithelioid change have been found in childhood infections and sudden infant death syndrome, and some evidence of a pathogenetic relationship between the two was found in this study. It is concluded that epithelioid change represents a definite pathologic alteration of germinal centers associated with total destruction of a whole population of cells. It probably represents an acutely acquired immunologic deficit and therefore may well contribute to the thus far uniformly fatal outcome of the associated diseases.

0722 ABDOMINAL TUBERCULOSIS: EXPERIENCES WITH 300 CASES. (Eng.) Bhansali, S. K. (81 Valentina Naoroji Gamadia Rd., Bombay 400 026, India). *Am. J. Gastroenterol.* 67(4):324-337; 1977.

Experience with 300 cases of surgically proven abdominal tuberculosis is reviewed. In 161 cases, the onset of illness was insidious; a definite lump was palpable in 96 of these patients. Acute manifestations were seen in 139 patients, with acute abdominal pain being the cardinal feature. Of the 139 patients who presented with acute manifestations, 92 presented with intestinal obstruction; roentgenologic studies detected fluid levels in the flat film of the abdomen in 91 of these 92 patients. Surgery was resorted to when complications were present, when diagnosis was in doubt, and when intrinsic intestinal disease was proved. Surgery was preceded by antituberculous drugs whenever possible. At surgery, the disease was found to involve the alimentary canal in 196 cases, with the small intestine being more frequently involved than the cecum and colon. In 104 cases, only the lymph nodes and/or the peritoneum were affected. Intestinal resection was performed in 101 cases. There were 22 operative fatalities, with 18 resulting after emergency operations. Significant postoperative complications were wound sepsis, bronchopulmonary complications, and fecal fistula. Death due to toxemia within 72 hr after surgery occurred in 15 patients. Positive histology was obtained in 229 cases, and 179 of these showed evidence of caseation. Caseation and peritoneal tubercles (103 cases) differentiated intestinal tuberculosis from Crohn's disease.

0723 A 44-YEAR EXPERIENCE WITH TUBERCULOUS ENTEROCOLITIS. (Eng.) Homan, W. P.; Grafe, W. R.; Dineen, P. (New York Hosp.-Cornell Medical Center, 525 E. 68th St., New York, NY 10021). *World J. Surg.* 1(2):245-250; 1977.

Thirty-one cases of intestinal tuberculosis, diagnosed between 1932 and 1975 at one hospital are reviewed, with particular reference to the impact of antituberculous chemotherapy on the outcome of the disease and to the role of surgery in diagnosis and treatment. The chest x-ray was 90% effective in demonstrating evidence of tuberculosis. However, since even on gross examination differentiation of intestinal tuberculosis from Crohn's disease or neoplasia may be impossible, surgical exploration is indicated when diagnosis is in doubt, and biopsy and culture of intraabdominal tissue should be performed for diagnostic purposes. The importance of instituting immediate therapy is emphasized by a 67% mortality rate in the prestreptomycin era and a 21% mortality rate in patients receiving appropriate drug therapy. Because of the success of chemotherapy, intestinal resection is seldom indicated, except in those cases in which scarring and stricture formation demand surgical relief of intestinal obstruction or in which the complications of fistula, hemorrhage, or perforation may occur.

0724 GASTRIC MUCOSA IN PATIENTS WITH RHEUMATOID ARTHRITIS TREATED WITH ANTI-INFLAMMATORY AGENTS. CLINICAL, RADIOLOGICAL, ENDOSCOPIC, PATHOLOGICAL AND ULTRASTRUCTURAL STUDY. (Fre.) Gaucher, A.; Serot, J. M.; Gaucher, P.; Rauber, G.; Grignon, G.; Pourel, J.; *et al.* (Hopital de Brabois, F 54500 Vandoeuvre-les-Nancy, France). *Nouv. Presse Med.* 5(41):2781-2784; 1976.

0725 THE DIGESTIVE ORIGIN OF Campylobacter SEPTICEMIAS. (Fre.) Butzler, J. P.; Derume, J. P.; Barbier, P.; Smekens, L.; Dekeyser, J. (Hopital Universitaire St-Pierre, 322, rue Haute, B-1000 Brussels, Belgium). *Nouv. Presse Med.* 6(12): 1033-1035; 1977.

0726 RESULTS OF A ONE-YEAR EPIDEMIOLOGICAL SURVEY ON DIGESTIVE TRACT CANCER IN GOLD COAST [Abstract]. (Fre.) Faivre, J.; Faivre, M.; Legoux, J. L.; Cueugnet, C.; Martin, F.; Michiels, R.; *et al.* (Hopital General, 21033 Dijon Cedex, France). *Gastroenterol. Clin. Biol.* 1(5):483-484; 1977.

0727 COMPARATIVE CHARACTERISTICS OF CORTICOSTEROIDS IN PATIENTS WITH DIFFERENT GASTROINTESTINAL TRACT CANCERS. (Rus.) Yelchits, T. V.; Mayevskaya, L. P.; Tarutinov, V. I. (Kiev Scientific Res. Inst. Roentgenology, Radiology and Oncology, Kiev, USSR). *Vrach. Delo* (3):40-42; 1977.

0728 COLON CARCINOMA--DURATION OF ANAMNESIS AND SURVIVAL TIME. (Ger.) de Heer, K.;

Eichfuss, H. P.; Vorwig, K. H. (Chirurgische Universitatsklinik und Poliklinik, Hamburg-Eppendorf, Martinistr. 52, D-2000 Hamburg 20, W. Germany). *Zentralbl. Chir.* 102(3):143-149; 1977.

0729 EOSINOPHILIC GRANULOMA OF THE GASTROINTESTINAL TRACT. (Ger.) Rendl, K. H. (Gefasschirurgische Abteilung, Landeskrankenanstalten, A-5020 Salzburg, Austria). *Wien. Med. Wochenschr.* 126(25/27):370-373; 1976.

0730 CLINICAL AND THERAPEUTIC STUDY OF CHOLERA AT DAHOMEY (OBSERVATION OF 133 CASES). (Fre.) Dournon, E. (No affiliation given). *Bull. Soc. Pathol. Exot.* 69(1):11-21; 1976.

0731 CLINICAL AND EPIDEMIOLOGICAL CHARACTERISTICS OF ACUTE INTESTINAL DISEASES INDUCED BY NONAGGLUTINATING VIBRIOS. (Rus.) Iakubov, O. Ia.; Dzhalalov, Kh. D.; Inzhevatova, M. V.; Mukhamedov, S. M.; Ziiaev, Sh. I.; Khusainova, S. A. (Uzbek Antiplague Station, Uzbek, USSR). *Med. Zh. Uzbek.* (11):58-59; 1976.

0732 CHARACTERISTICS OF THE CLINICAL COURSE AND DIAGNOSIS OF INTESTINAL E. coli INFECTION IN CHILDREN. CURRENT STATUS. (Rus.) Timofeeva, G. A.; Speranskii, N. P.; Gusarskaia, I. L. (Leningrad Scientific Res. Inst. Childhood Infections, Leningrad, USSR). *Pediatriia* (7):34-37; 1976.

0733 EFFECT OF BRAN ON BILE SALT METABOLISM IN CONSTIPATED PERSONS [Abstract]. (Fre.) Audigier, J. C.; Herr, P.; Minaire, Y.; Mallein, R.; Lambert, R. (INSERM U-45, Hopital Edouard-Herriot, 69374 Lyon Cedex 2, France). *Gastroenterol. Clin. Biol.* 1(4):396-397; 1977.

0734 DUPLICATION OF THE GASTROINTESTINAL TRACT. LITERATURE REVIEW AND CASE REPORT. (Ger.) Krenn, R. (Landeskrankenhaus Klagenfurt, Kinderchirurgische Abteilung, St. Veiter Strasse 47, A-9010 Klagenfurt, Austria). *Wien. Med. Wochenschr.* 126(20/22):302-304; 1976.

0735 COMPARATIVE EFFICIENCY OF DIFFERENT METHODS OF TREATMENT OF PATIENTS WITH ACUTE DYSENTERY. (Rus.) Gebesh, V. V.; Yanushevskaya, R. I.; Babak, V. I.; Kiriyenko, M. S.; Lysak, L. I. (Kiev Scientific Res. Inst. Infectious Diseases, Ukrainian SSR Ministry of Public Health, Kiev, USSR). *Vrach. Delo* (5):137-141; 1977.

0736 CONCENTRATION OF CORTISOL, GASTRIN, INSULIN AND GROWTH HORMONE IN THE BLOOD SERUM OF PATIENTS WITH CHRONIC ENTEROCOLITIS. (Rus.) Ivanov, S. G. (USSR Ministry of Public Health, Moscow, USSR). *Ter. Arkh.* 49(5):79-82; 1977.

0737 DIGESTIVE TRACT HEMORRHAGES DUE TO ACUTE DRUG-INDUCED ULCER: PROGNOSIS AND TREATMENT [Abstract]. (Fre.) Franco, D.; Murat, I.; Deporte, A.; Bismuth, H. (Hopital Paul-Brousse, avenue Paul-Vaillant Couturier, 94800 Villejuif, 14, France). *Gastroenterol. Clin. Biol.* 1(5):482; 1977.

0738 CLINICAL ASPECTS OF THE NONTUMOROUS DISEASES OF THE SALIVARY GLANDS. (Ger.) Haubrich, J. (Univ.-Hals-Nasen-Ohrenklinik, Geiststr. 5/10 D-3400 Gottingen, W. Germany). *Arch. Otorhinolaryngol. (NY)* 213(1):1-59; 1976.

0739 GASTROINTESTINAL HEMORRHAGES AS COMPLICATIONS OF MYOCARDIAL INFARCTION. (Rus.) Belousova, I. M. (6th Clinical Hosp., USSR Ministry of Public Health, Moscow, USSR). *Vrach. Delo* (5): 16-21; 1977.

0740 IMMUNOCYTES AND IMMUNOGLOBULINS IN THE DIGESTIVE TRACT DURING PRIMARY IMMUNE DEFICIENCY IN ADULTS [Abstract]. (Fre.) Matuchansky, C.; Galian, A.; Goldfain, D.; Rambaud, J. C.; Modigliani, R.; Bernier, J. J. (Hopital Saint-Lazare, 107, rue du Faubourg-Saint-Denis, 75010 Paris, France). *Gastroenterol. Clin. Biol.* 1(5): 485; 1977.

0741 PRESSURE TEST FOR DETECTION OF MICROPERFORATIONS. CONTRIBUTION TO CHOLEPERITONEUM WITHOUT PERFORATION. (Ger.) Walzel, C. (Chirurg. Abteilung, Krankenhaus der Barmherzigen Bruder, Grosse Mohrengasse 9, A-1020 Vienna, Austria). *Chirurg* 47(6):345-347; 1976.

0742 PROBLEMS OF Saccharomycopsis IN THE GASTROINTESTINAL TRACT. (Ger.) Knoke, M.; Bernhardt, H.; Schwesinger, G. (Med. Klinik und Poliklinik, Ernst-Moritz-Arndt-Universitat, Friedrich-Loeffler-Str. DDR-22 Greifswald 23, E. Germany) *Dtsch. Z. Verdau. Stoffwechselkr.* 36(2):69-71; 1976.

0743 SALIVARY GLAND TUMORS OF UNUSUAL LOCALIZATION. (Ger.) Kecht, B. (Untere Donaulande 26, A-4020 Linz, Austria). *Wien Med. Wochenschr.* 126(25/27):363-365; 1976.

0744 ABDOMINAL TRAUMA: INJURIES OF THE DIGESTIVE TRACT AND RETROPERITONEAL ORGANS. (Ger.) Allgower, M. (Chirurgisches Departement, Kantonsspital, CH-4031 Basel, Switzerland). *Helv. Chir. Acta* 44(1/2):63-72; 1977.

0745 CHARACTERISTICS OF THE CLINICAL COURSE OF MODERN TYPHOID FEVER. (Rus.) Shugailo, V. T.; Kimelblat, M. A.; Yaremchuk, Y. A.

(Ivano-Frankovsk Medical Inst., Ivano-Frankovsk, USSR). *Vrach. Delo* (3):140-142; 1977.

0746 VALUE OF THE SECRETIN TEST IN THE DIAGNOSIS OF ZOLLINGER-ELLISON SYNDROME, PROGRESSIVE DUODENAL ULCER WITH BASAL GASTRIC ACID HYPERSECRETION AND IN RECURRENT ULCER FOLLOWING SURGERY FOR DUODENAL ULCER [Abstract]. (Fre.) Mignon, M.; Accary, J. P.; Bonfils, S. (Hopital Bichat, 75018 Paris, France). *Gastroenterol. Clin. Biol.* 1(5):491; 1977.

See also, 0093, 0218, 0238, 0246, 0286, 0288, 0294, 0296, 0302, 0316, 0337, 0388, 0419, 0421, 0452, 0457, 0503, 0510, 0521, 0532, 0652, 0713, 0768.

GENERAL

Parasitology

0747 VISUALIZATION OF THE AMEBIC ABSCESS OF THE LIVER WITH PERFUSION OF DIATRIZOATES. (Fre.) Limbos, P.; de Bergeyck, E. (Institut de Medicine tropicale d'Anvers, Antwerp, Belgium). *Acta Gastroenterol. Belg.* 40(3/4):159-166; 1977.

0748 AMEBIASIS. PART 2. (Ger.) Werner, G. T.; Stickl, H. (Bayerische Landesimpanstalt Munchen, Munchen, W. Germany). *Med. Klin.* 71(51/52):2254-2259; 1976.

0749 HYDATID CYSTS OF THE LIVER AND ECHOGRAPHY. (Fre.) Pietri, H.; Roussille, M.; Duquesnel, C. (No affiliation given). *J. Radiol. Electrol. Med. Nucl.* 57(8/9):606-607; 1976.

0750 HYDATID DISEASE DUE TO Echinococcus multilocularis: ANALYSIS OF 43 CASES TREATED AT NANCY [Abstract]. (Fre.) Gabriel, A.; Gaucher, P.; Bigard, M. A.; Penin, F.; Rothhahn, B.; Fays, J.; *et al.* (No affiliation given). *Gastroenterol. Clin. Biol.* 1(4):398-399; 1977.

0751 HYDATID CYSTS OF THE LIVER IN CHILDREN. REPORT OF 40 CASES. (Fre.) Saied, H.; Paley, D.; Dupre, A.; Chantereau, M.; Lacki, W. (Hopital des Enfants, Tunis, Tunisia). *Ann. Chir. Infant.* 18(1):58-61; 1977.

0752 SURGICAL TREATMENT OF HYDATID CYST OF THE LIVER. (Fre.) Maillet, P. (No affiliation given). *J. Med. Lyon* 57(1303):347-352; 1976.

0753 INFESTATION WITH Lamblia intestinalis. (Pol.) Ananko, J.; Jankowska-Bartel, M.; Porada, D. (Wojewodzki Szpital Zespolony, ul. Tochtermana 1., 26-600 Radom, Poland). *Wiad. Lek.* 30(6):429-432; 1977.

0754 TREMATODES IN THE BILE DUCTS. (Ger.) Dippon, R.; Widmer, A. (Chir. Klinik, Kantonsspital, CH-5000 Aarau, Switzerland). *Helv. Chir. Acta* 43(5/6):553-555; 1976.

0755 GIARDIASIS IN INFANCY AND MALABSORPTION SYNDROME [Abstract]. (Eng.) Fagundes Neto, U.; Wehba, J.; Patricio, F. R. S.; Machado, N.; Silvestrini, W. S. (No affiliation given). *Arq. Gastroenterol.* 14(3):159-160; 1977.

0756 FLUORESCENT ANTIBODY STUDIES IN GIARDIASIS [Abstract]. (Eng.) Wright, S. G.; Moody, A. H.; Tomkins, A. M.; Ridley, D. S. (Hosp. Tropical Diseases, London, England). *Gut* 18(11):A986; 1977.

See also, 0233, 0285, 0419, 0444, 0468, 0490, 0492, 0498.

GENERAL

Regional Enteritis

0757 INTRAMURAL GANGLION CELL DEGENERATION IN INFLAMMATORY BOWEL DISEASE. (Eng.) Oehmichen, M.; Reifferscheid, P. (Institut fur Hirnforschung, Universitat Tubingen, Belthlestrasse 15, D-7400 Tubingen, W. Germany). *Digestion* 15(6):482-496; 1977.

Comparable histologic alterations of intramural

nervous tissue were reported in various diseases of the small and large intestine. In six patients, an intestinal obstruction led to secondary intramural inflammation. The obstruction was induced by the spastic segment in Hirschsprung's disease (3 cases), by rectal atresia (2), and by postoperative ischemic damage to the intestinal wall (1). In four other patients, the inflammation was predominant, and it was due to necrotizing enterocolitis (1 case), membranous enteritis (1), and Crohn's disease (2). In all cases, various degrees of inflammatory lymphocytic infiltration were observed proximal to the obstruction. Other common alterations were splitting of nerve fibers and deteriorations of nerve cells (degenerative alterations) in the plexus myentericus, as well as hyperplasia and hyperchromasia of the ganglion cells, Schwann cell proliferation, and fibrosis in the nerve fibers (reactive alterations). Until now, these alterations were only observed in Chagas' disease and in ulcerative colitis, especially when these conditions were complicated by toxic megacolon. The neurodegenerative alterations in these diseases may be toxically induced.

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★U.S. GOVERNMENT PRINTING OFFICE: 1979—281-227:1

DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
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DHEW Publication No. (NIH) 79-66